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NOTES ON 50 CASES OF GASTRIC ULCER.

Thesis for the Degree of M.D.,

by

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NOTES ON 50 CASES OF GASTRIC ULCER.

Definition.

Gastric Ulcer is an affection of the stomach beginning in the mucous membrane, and sometimes extending through all the coats of that organ by a process of the nature of a necrosis.

Etiology.

1. Frequency of Incidence.

Cases of gastric ulcer have been recorded from the 16th. century, and even earlier, but it was not until Matthew Baillie[1] in 1793, and later Cruveilhier[2] in 1829, gave an accurate anatomical and clinical description that the disease was established as a definite entity.

Statistics are, on the whole, of doubtful value.

(a) Records of post-mortem examinations are often fallacious, since small open ulcers are frequently not discovered, and cicatrices of healed ulcers are overlooked. The following figures from results of autopsies shew considerable variation. In London, Fenwick[3] noted the occurrence of gastric ulcer in 4.6% of 10,000 autopsies; in Berlin, in 2.7%[4]; in Munich in 1.2% of 3,500[5]; in Kiel, 8.3%; in America,

1.32% of 10,841[6]; and in Europe, 8.54%.

(b) Clinically the same variance in the records is observed.[7] In Edinburgh the percentage formed is 2.2% of all admissions; in London, 0.82%; in Berlin, 1.33%; in America, 0.57%.

2. Age.

Gastric ulcer is most frequent between 20 and 30 years of age in the female, and between 30 and 50 in the male. Combined statistics shew thatt the average for females is 27.1 years of age, and for males 36.7 years.

In my 50 cases there were 33 females, and the age incidence is best shewn in the table.

	Under 20 years.	Between 20 & 30.	Between 30 & 40.	Between 40 & 50.
Number of Cases	3	15	10	5

The commonest age was 25. This corresponds to what is generally stated.

There were 17 males in my series, with the age incidence tabulated as follows.

	Under 30 years.	Between 30 & 40.	Between 40 & 50.	Over 50.
Number of Cases	6	8	1	2

The commonest age was 39 years. This is a little over what is generally mentioned.

Cases of gastric ulcer have been observed in infants, and occasionally after birth. Martha Wollestein found 5 cases of gastric ulcer out of

390 autopsies at the Babies' Hospital,New York. The earliest age in my series was,in the female, 15 years,and in the male,26 years.

After 70 years of age,death from gastric ulcer is rare,though it may occur,as in the cases reported by Weber,Sedgwick,Fenwick,etc.. The latest age in my 50 cases was 48 years in the female,and 54 in the male.

3.Sex.

Statistics on this point shew that the ulcer is more frequent in females than in males. The ratio generally accepted,as determined from autopsies,is 2 to 1. At Manchester the incidence is 2.5 to 1. Other observers give the following: 1.7 to 1(Habershon)[9]; 134 to 126(Riegel); 3 to 2(Welch,the Fenwicks). Lebert,[10] on the other hand,found it more common in males at Breslau. The proportion from my cases is 33 females to 17 males. This agrees with what is usually stated, as it is practically 2 to 1.

Clinical records shew an even higher ratio in females; for example,Fenwick found the incidence to be in the proportion of 3 females to 1 male; Lebert found it to be 3.5 to 1. The Fenwicks found from post-mortem examinations that acute ulcers were confined more especially to females,whereas the chronic ulcers were found in males.

The frequency of the disease is greater in Europe than in America; in Britain it is commoner in Edinburgh than in London.

4.Influence of occupation as a causative factor can be seen from statistics. These shew that cooks are affected most,possibly because of tasting food when it is very hot. Out of 33 females in my list,there was only one cook and one confectioner. In the Midlands,the occupation in which ulcer is found most frequently is that of domestic servant; as many as 22 were recorded by me. Workers in metals, and in glass and porcelain factories,who swallow irritating dust particles,are thought by some to be more liable. I can record only 10 whose work is connected with metal,glass or porcelain. I had one case of a barmaid,and one of a barman; neither was teetotal. Occupations in which there is direct mechanical pressure on the epigastrium(such as those of tailors,shoemakers and weavers) and the wearing of tight corsets are said to exert an influence in the production of ulcer. I had but one case of the occupations just stated,and that was a tailoress. There were three cases in which lifting heavy weights may have been a cause,namely,those of a market porter,carman and hotel porter. There was one policeman in my list.

5.The Influence of Habits has been regarded as a causative agent. It is said to be very common among those who take insufficient or badly pre-

pared food. In the histories obtained by me, the commonest cause seemed to be excessive tea-drinking, with bread and butter. These patients would rather take tea than cook or eat meat.

6. Traumatism as a cause of ulcer. This may be external or internal.

(a) External. The injury may be a blow or compression from a cobbler's last. Ackermann[11] observed 16 cases of post-traumatic ulcer. Ebstein[12] mentions two cases of gastric ulcer produced by lifting weights. Other instances are mentioned by Pauly, Richardière and Dr. Moore. Burns of the skin are indirectly related to gastric ulcers.

(b) Internal. The swallowing of hot foods, corrosives, foreign bodies and irritants may produce gastric ulcer.

7. Heredity. may be a predisposing factor in the production of an ulcer, though most authors make no mention of it. There are instances where ulcer has occurred in more than one member of the same family. In two instances mother and daughter were affected, and in one instance father and daughter. In two instances, two sisters were affected. The Fenwicks found that the disease was present in one of the parents in about 3%, and in some other member of the family in nearly 5%. I had 7 instances in the series of 50 cases shewing that one parent or relative was affected in the same family

In six of these seven cases the patient was a female, in one a male. In three instances one parent - the mother - had had ulcer of the stomach. One mother had died from a haematemesis. There was no case of the father or both parents being affected. There were two instances in which the patient had two sisters affected; in one case one of these sisters had had an operation performed for the ulcer. The remaining two cases had one sister affected in a similar manner.

8. There are certain diseases that predispose to the production of an ulcer, notably, chlorosis, anaemia, arteriosclerosis, tuberculosis, endocarditis, scurvy, chronic Bright's disease, syphilis, previous gastric ulcer.

Chlorosis is frequently associated with amenorrhoea, which is to be regarded as a phenomenon of the blood condition, and not as a causal agent in the production of an ulcer. Amenorrhoea preceded the ulcer in 19% cases (Fenwick). Riegel regards chlorosis as primarily inducing hyperacidity, which in turn helps to cause the ulcer.

In my list anaemia predominated amongst the females, but was absent in the males, except to a slight extent after a haematemesis. Anaemia was complicated by other diseases, having followed some of the acute fevers, Diphtheria (one case), Rheumatic

Fever(4 cases),Scarlet Fever(one case). Associated with anaemia was a well-marked history of chronic constipation,and for a certain period amenorrhoea. Out of my 50 cases there were 12 instances of anaemia,with a history of having attended the out-patient department for a considerable time. This gives 24% for my series of 50 - 5% higher than Fenwick's figures.

Arteriosclerosis and Endocarditis seem at times to be causes. Howard mentions that 48% of his cases had sclerosed vessels. There was no case of arteriosclerosis amongst my female patients,though it was present in 5 cases of the 17 males. Endocarditis was present in four cases - all females-the mitral valve in each case being the one primarily affected. Two of these were found to have an escape murmur at the tricuspid valve.

Tuberculosis in the lungs as a definite cause of gastric ulcer has not been met with,though it is frequently associated with it,as the two are concomitant in 17% cases. On the other hand,the percentage of pulmonary tuberculosis and gastric ulcer at the Brompton Hospital is 0.9. I had no case in my series with pulmonary tuberculosis. The suggestion put forward is that the ulcer may be a portal of infection by the tubercle bacillus. Specific ulceration by the tubercle bacillus is occasionally met with in the stomach.

Chronic Bright's Disease is frequently associated with haemorrhagic erosions and indolent ulcers of

the stomach. I had two cases - both males - in which chronic nephritis and alcoholism were present. Syphilis may cause gastric ulcer as a result of interstitial and blood changes, e.g., necrosis of the mucous membrane as a result of syphilitic endarteritis; or through the breaking down and softening of a gumma; or even as a consequence of cachexia. There was one case (a male) who gave a history of luetic infection.

Previous gastric trouble is difficult to estimate, though Mathieu and Galliard regard gastritis - especially if associated with hyperacidity - and the existence of localised plaques, as a most important predisposing cause. This is supported certainly from the evidence of the past histories of the patients under my care. There were 28 instances out of 50 that gave a history of old gastric trouble - over 50% of the total. Of these 26, 15 were females and 13 males. The periods over which the chronicity of the disease extended varied. The longest was in the case of a man with a 16 years' history of chronic indigestion. One woman had a 12 years' history; two men a 10 years' history; one man an 8 years' history; two other men 7 years'; one woman and one man each recorded 6 years' gastric trouble. Two women recorded a five years' history, and four years' history was given by both one female and one male patient. One woman and one man each recorded three years' gastric [illegible] symptoms; whilst two years'

history was obtained from six women and two men. Two women gave 1½ year's and two men one year's chronicity. With few exceptions the history obtained extended over less than six months. The shortest period of gastric ~~te~~ trouble that was present in any of my cases was 14 days. Some of the patients with the longer periods of disease gave histories of more than one haematemesis, or had even undergone an operation. The following table summarises these statements.

	14 days	under 6 months	1 year	1½ years	2 years	3 years	4 years	5 years
No. of cases	1	22	2	2	8	2	2	2

	6 years	7 years	8 years	10 years	12 years	16 years
No. of cases	2	2	1	2	1	1

Morbid Anatomy.

Ulcers having the characteristic appearances of gastric ulcer are found where the gastric juice flows; therefore they are to be seen in the stomach, in the duodenum, in the lowest part of the oesophagus, and in the jejunum after gastroenterostomy.

There are two types _ acute and chronic - according to rapidity of production.

Number. Ulcers are usually single; occasionally more than one is found. Brinton noted as many as five ulcers in two cases, and more than five in four cases. The Fenwicks found the percentage of single and multiple ulcers as follows: One ulcer in 80.5: 2 ulcers in 12.1: 3 in 3.1: 4 or more in 4.26. In short, the lesion is multiple in about one-fifth of all cases.

Of those of my cases that admitted observation, either from operation or from post-mortem examination, I found that the lesion was multiple in three patients, two of these shewing three ulcers, the remaining one, two ulcers. The ratio of multiplicity of lesion is 3 to 50, or 6%. The lesion was single in twelve cases, or 24%.

Fenwick further states that the acute ulcer was multiple in 54%, the chronic form multiple in 13%. There was but one case of the three quoted above as shewing multiplicity that was acute; the other two were chronic and multiple. Of the 12 cases shewing single ulcers, 4 were acute and 8 chronic, or 8% and 16% respectively.

Situation. The lesser curvature is the commonest seat of ulceration, on either its anterior or posterior surface. Details of Brinton's (13) statistics shew the posterior surface of the stomach to be the commonest site. Welch (14) and the Fenwicks state the lesser curvature to be the most frequent position of the ulcer, especially towards the pylorus. The chronic ulcer is more often found at the pylorus, whilst the acute is situated near the pylorus. In all my 15 cases where the position of the ulcer was ascertained, I found that it was within two inches of the pylorus, but on different walls. For instance, there were six located on the lesser curvature, and of these six only one was acute, the other five being chronic. There were eight on the posterior wall, six being

chronic ulcers. One chronic ulcer situated near the pylorus had extended completely round the lumen of the stomach, being an example of the annular type. There were three ulcers situated on the anterior wall, two being chronic and one acute. So that, from the above, my cases shewed the most frequent site to be the posterior wall within two inches of the pylorus, the next in frequency being the lesser curvature, where the ulcer was also within two inches of the pylorus. In ~~the~~ two instances the stomach shewed the scar of a healed ulcer, in the presence of an acute ulcer in one case and of a chronic ulcer in another case.

Macroscopic Appearance. Shape. Acute ulcers are punched out in appearance: usually round: sometimes oval: the edges are sharply cut out and limited: the floor is smooth and firm, though occasionally irregular. Small haemorrhages are usually to be found in the neighbourhood. The depth of the ulcer varies, sometimes reaching to serous coat, having ulcerated through the intervening layers. This ulcer is usually funnel-shaped. The less acute and chronic ulcers are terraced or shelved and frequently obliquely directed. This fact was pointed out first by Orth and Virchow as being due to the line of distribution of the blood-vessels. Fenwick is of opinion that the differing contractility of the several coats of the stomach produces the funnel-shaped variety.

I had three cases of acute ulcers,two of which were seen at autopsy. The third was successfully operated on. The description of the two found at the autopsy tallies with the above very closely. The first case,that of Arthur Horton,admitted into the General Hospital,Birmingham,was the result of a fatal haematemesis,and was described as an ulcer lying on the anterior surface of the stomach about 1½ inches from the pylorus. The ulcer was deep and funnel-shaped,measuring nearly one square inch - extremely large for an acute ulcer - and had penetrated the mucous,submucous and muscular coats to the peritoneum,which formed the floor of the ulcer. The peritoneum was injected and neither puckered nor thickened. At the bottom of the ulcer was the opening of an artery of considerable size. The second case was very similar and was the result of the death of Charlotte Hardwick in the General Hospital,Birmingham,from a fatal haematemesis. This ulcer was punched out,but irregular in shape,and likewise penetrated to the peritoneum in a funnel-shaped manner. The peritoneum was puckered,and recent scanty adhesions were found between it and the anterior surface of the pancreas,the ulcer being located on the posterior wall near the pylorus. The mouth of a vessel of large size was found at the base of the ulcer.

The third acute case was that of a patient,

William Poynter,who,whilst in the St.Giles Infirmary,London,suddenly developed a subphrenic abscess,with the formation of gas in the upper abdomen. This case was operated on successfully by Mr.Clayton Greene,who found an acute perforating ulcer,which had penetrated all the coats of the stomach as well as the serous. This ulcer was situated on the anterior wall of the stomach,and was funnel-shaped,with a punched out margin. There was injection of the neighbouring portion of mucous membrane.

A chronic ulcer is usually larger,irregular and oval, shewing signs of old cicatrisation. Its edges are usually hard and thickened. The floor is smooth or irregular and hard,and formed of dense fibrous tissue. The neighbouring parts shew some chronic inflammation,and usually a varicose condition of the blood-vessels. Adhesions to the surrounding tissues may occur. The size varies according to whether the ulcers are acute or chronic. Acute ones are small,measuring about half an inch. Cruveilhier described one as measuring $6\frac{1}{2}$ X $3\frac{1}{2}$ inches. Of this type I had twelve cases,two of which were examined in the post-mortem room. The other ten were operated upon. I shall not describe each one in detail at this juncture,but take the case of Elizabeth Green,who was in the General Hospital,Birmingham,as an example. A large,

irregular ulcer was found lying on the lesser curvature,about two inches from the pylorus. It extended on to the upper part of the anterior wall, and to a greater extent on to the posterior. The edges were hard. The wall of the stomach was in part eroded completely,the viscus being adherent to the diaphragm and to the under surface of the left lobe of the liver,both of which assisted in forming the floor of the ulcer. Elsewhere in the floor, which was still formed by the coats of the stomach, eroded blood-vessels were seen. This was a case, post mortem,that died from a ~~inguinal~~ suppurative peritonitis,not the result of surgical interference, as the operation wounds were quite healthy. In the other cases there was considerable induration and thickening,with perigastric adhesions;but these will be described later.

Microscopic Appearance. The changes in acute ulcer are those of necrosis,in chronic ulcer those of inflammation. The gastric glands at the edge of the ulcer are tortuous and many are converted into cysts. There is now no distinction between parietal and central cells. Interglandular tissue is thickened, and there are signs of cell proliferation and infiltration,with the formation of fibrous tissue. Muscle cells are replaced by fibrous tissue cells; the greater the chronicity,the greater and denser is the formation of fibroblasts. Marked inflammatory

changes in and around the blood-vessels are seen, with the production of endarteritis and sometimes thrombosis. Between the several layers of the stomach coat, there is well-advanced cell infiltration.

Healing and cicatrisation are common, and commence from the sides and floor of the ulcer. Puckering and contraction occur. New gland tubules are formed, though small and atypical, usually with cylindrical cells arranged round a lumen. There is generally no secretion from these cells. The healing process is slow and irregular, and may leave chronically inflamed areas which have a tendency to bleed. In this process of repair much deformity may ensue, depending on the position of the ulcer and extent of the adhesions. The deformities comprise hour-glass contraction, pyloric stenosis with subsequent gastrectasis, diverticula and irregularities. There may be no obstruction. Grünfeld estimates that 20% of all cases cicatricise.

In the process of inflammation, the ulcer forms adhesions, at first fibrinous, later fibrous. The frequency with which adhesions are formed has been estimated by von Jaksch as being 40%, by Lebert at 42%, and by the Fenwicks at 46%. The viscera most frequently implicated in the adhesions are pancreas alone to the extent of 40%, liver alone 26.8%, pancreas and liver 8.1%, colon 5.7%, and three or more organs 12.2%. The union of the

stomach to the other viscera is effected by fibrous tissue,but not infrequently the ulceration extends through this bond to the attached viscus, which may now form the floor of the ulcer. The pancreas,liver or spleen has formed the floor of the ulcer.

In regard to my cases,adhesions were formed in 7 of the 15 cases in which confirmation could be obtained,shewing a percentage of just under 50,which approaches Fenwick's figures. The viscera implicated were pancreas and liver(two cases),pancreas only (one case),diaphragm and liver(one case), transverse colon(one case),omentum(one case),stomach bed (one case).

If the fibrous tissue bond is not firm, perforation of the ulcer may occur,and the subsequent result depends on the site of the ulceration. If the ulcer perforates into the general peritoneal cavity,the gastric contents escape,giving rise to peritonitis. The extent of this depends on the size of the opening. If it is small,fresh adhesions may form and so localise the peritonitis,possibly with the production of a subphrenic abscess; if the opening is larger,an extensive general peritonitis will result. The perforation may take place into an adjoining viscus,with the production of a fistula. This occurs between the stomach and colon or stomach and duodenum. Perforations are

most common when the ulcer is situated on the anterior wall of the stomach. Brinton states that this is so in 70%. Of the cases coming under my notice, three perforated; the ulcer was situated on the anterior wall in two cases, with the production of subphrenic abscesses, and the third case was located on the lesser curvature. The Fenwicks make a reference to the fact that acute ulcers perforate the anterior wall near the lesser curvature, whilst the chronic ulcers perforate the posterior wall in the pyloric portion.

From what has been stated previously, it is seen that ulcers as they progress tend to form adhesions or perforate. They also tend to necrose the blood-vessels, with the production of haemorrhage, which may or may not cause death. I had three cases of fatal haematemesis, one resulting after the operation performed for its cure. In each case the vessel eroded was easily seen and allowed a fairly large bristle to be passed down its lumen. The more severe haemorrhages result from erosion of the wall of an artery or vein, especially branches of the coronary, gastro-epiploic, and sometimes splenic arteries. Less severe haemorrhages occur from the capillary vessels, and may be so slight as to be seen with great difficulty by the naked eye either in the vomit or in the faeces. As has been mentioned, a cicatrix is formed in the process of healing. If the ulcer has penetrated to some depth,

a dense,white,firm cicatrix,slightly depressed,is seen. If the ulcer has been extensive,much deformity will be produced from the contraction of the fibrous cicatrix. The most frequent deformity caused thereby is Pyloric stenosis,with subsequent dilatation of the stomach(Gastrectasis). This occurs in from 16% to 20% of all chronic ulcers. It should be stated that the stenosis may be in part due to spasm of the pylorus,or even kinking at the pyloro-duodenal junction. There were in my list seven cases of dilated stomach of varying degree,the result of contraction of the ulcer near to the pyloric orifice.

Another result of cicatricial contraction is Hour-glass Stomach. This occurs when the ulcer is situated on the lesser curvature and extends transversely to the long axis of the stomach. This constriction thus divides the stomach into two cavities, the larger usually corresponding to the fundus,the smaller to the pyloric portion. The communicating opening may be as small as the diameter of a pencil, or yet again,as large as to allow two fingers to pass through. I have no cases illustrative of this condition.

Irregular Deformity occurs when the healing ulcer,situated on the lesser curvature,contracts and approximates the two orifices.

Contraction of the Stomach arises from a diffuse perigastritis,by cicatrisation of a

chronic ulcer in the neighbourhood of the cardiac orifice.

Diverticula, or localised bulging of the stomach wall, occur as a result of cicatrisation. The ulcer is frequently situated near the pylorus.

Pathology.

The question arises now, "What is the exact cause of gastric ulcer?" There is probably more than one cause operating. The literature on this subject is rich in theories, and experimental and theoretical observations have been made in several directions, by many investigators. It seems natural to suppose that the stomach, which is the only organ subject to this type of ulcer, is corroded by the action of the gastric juice. In sequence to this supposition, one asks, "Why does not the gastric mucosa digest itself under normal conditions?" This does not occur during life, but the digestion of the mucosa by the gastric juice does occur after death, as John Hunter pointed out in 1786.

I. Circulatory System in Relation to Gastric Ulcer.

One theory to account for this condition is that, during life, alkaline blood is continuously flowing through the gastric mucosa, consequently neutralising the acid juice. If the circulation is impeded in any way, auto-digestion of that affected portion occurs. Virchow in particular upheld the circulatory theory. He demonstrated that embolism, thrombosis, trophic disturbances of the

vessel walls,and varicose dilatations are the most prolific causes in producing the haemorrhagic necrotic origin of gastric ulcer. This theory was advanced in 1855. Against this view are the following objections,namely:-

(a) The gastric arteries are not terminal.

(b) Emboli are not always found.

(c) Ulcers occur most frequently when no cause of embolism exists,and in young people.

(d) Thrombosis is most probably a result,rather than a cause of the ulcer.

Wilkie (15) as recently as 1910 demonstrated that retrograde venous embolism is a determining factor in the production of acute ulcer. Most observers agree on this point,namely,that interruptions in the circulation of the blood in circumscribed areas constitute the chief cause of gastric ulcer. The local nutrition of the gastric mucosa is interfered with,and this renders that part easy of digestion by the juice.

II. Relation of Hyperacidity to Gastric Ulcer.

Another theory was promulgated by Riegel, who believed that there was hyperacidity of the gastric juice. Pavy,in addition,stated that there was reduced alkalinity of the blood. This view is used as a basis for experimental investigation, though it does not explain the cause. It is more frequently found that the percentage of HCl.

is normal,and not increased. Ewald,from his experiments,found hyperacidity in 34.1% and normal acidity in 56.8%. Wirsung found hyperacidity in 42.7% and normal acidity in 55.5%. Howard found hyperacidity in 17.6% only,and normal acidity in 56.0%. On the other hand,Einhorn in America,Robin in France and Fenwick in England found hyperacidity in most of their cases. Fenwick's results amounted to 75%. These variations may be explained by the fact that there is some difference of opinion as to what constitutes hyperchlorhydria; also the means of testing and colour tests are not uniform.

It is certain that gastric ulcers occur only where there is gastric juice,though hyperacidity is not essential to their formation. It is common to get an acidity. Again,hyperacidity is found in the absence of ulcer,and is insufficient by itself to produce it. The fact is,hyperacidity may induce an ulcer when there is some injury to the mucosa,and by virtue of the acid the ends of the vessels are constricted,producing an ischaemia and so necrosis.

It had been stated by Pavy that the alkalescence of the blood protected the stomach from the action of the juice. Therefore,if this be correct,diminished alkalinity of the blood favours auto-digestion,because the acid is not neutralised. (16)
Samelson,by his experiments,pointed out that the alkalinity of the blood did not protect the mucosa. In fact,the chemical composition of the blood is

unimportant. Besides, the alkalinity of the blood is so small that it can hardly be considered in relation to the acidity of the gastric juice. It might be adduced, in consequence of the reduced alkalinity of the blood in chlorotics, that alkalescence of the blood prevented digestion by the acid juice. The explanation seems to be that in anaemia there is retardation in the healing of the ulcer. These statements have been verified by experiments of Quincke[17] and Daettwyler and Silbermann[18]. The main reason for the absence of digestion of the gastric mucosa by the juice is undoubtedly the vital resisting power of the tissues. This protective influence of living tissues, especially epithelial structures, was termed by Hunter "the vital principle." This protection has been ascribed to an alkalinity of the blood in the gastric mucosa.

III. Bacterial Necrosis as a Cause of Ulcer.

Gastric ulcers have been found in pyaemia, septicaemia and other infective diseases. These ulcers are more of the nature of haemorrhagic erosions, on the borders of which organisms have been found by Boettcher[19], Letulle and Turck[20] and Stokes. Experimental injection of Staphylococcus pyogenes and B.coli communis into the stomach or peritoneum has produced ulcerations. S.Martin believes that if bacteria lodge in the mucous follicles of the stomach, they will produce necrosis. The more

frequent occurrence of ulcers towards the pylorus is explained by the fact that there are no glands secreting HCl.,which in consequence is unable to exert its powerful antiseptic action. In support of this there is one clinical fact,namely,the prevalence of constipation in young anaemic women,coexisting with gastric ulcer. It is concluded that organisms are developed in the bowel as a result of prolonged absorption,and these are carried in the blood and lymph streams,forming emboli in gastric vessels,with subsequent necrosis. The above remarks shew some pathological evidence that necrosis can be brought about by bacteria.

IV.Gastrolysis as a Cause of Ulcer.

It has been shewn experimentally by C.Bolton (21) that the cells of the gastric mucosa in their destruction are capable of producing certain organic substances which have a specific cytotoxic action. This specific action is gastrolytic and haemolytic to the cells in the stomach,and in the presence of the gastric juice causes necrosis of the mucosa. It was demonstrated by the same experimenter that blood poisons circulating inflict slight damage to the cells of the gastric mucosa and the action of the juice in these cells starts the necrosis. He further proved that hyperacidity alone would not cause necrosis,but it would do so in the presence of another poison.

To summarise these findings on Etiology,it

can be stated that under normal conditions the gastric juice has no solvent action on the healthy gastric mucosa,unless a blood poison is circulating and injuring the cells at the time. There is in the majority of cases some local injury or death,before the juice can exert its autolytic action. The local injury may be traumatic,chemical,etc.,associated with hyperacidity or invasion of organisms. The local death is caused by thrombosis of the vessels, or embolic processes derived from heart or pulmonary disease,portal congestion,bacteria,or varicosity of the stomach vessels.

Symptomatology.

Though symptoms are often so dramatic and obvious,yet,on the other hand,there may be none at all. Frequently this is seen in the post-mortem room, in bodies in which gastric ulcers have been found,which yet during life gave no symptoms referable to the existence of an ulcer. Stoll found ulcers were latent in 27%; Savariand in 20%. Sometimes the first evidence of the existence of an ulcer is a profuse and possibly fatal haemorrhage; yet again,a sudden perforation may produce an extensive peritonitis; or perhaps the production of a subphrenic abscess may be the first sign. I have one case shewing latent symptoms.

Though the symptoms may be latent,there are usually well recognized features which,if all are

present,leave no doubt in the physician's mind. The ulcer may produce ill-defined symptoms at first,until certain others become outstanding. In the initial stage there is probably the complaint termed by the patient "dyspepsia." By this he means a sense of fulness and weight,accompanied by gaseous eructations and a feeling of burning or gnawing. These symptoms gradually subside and leave the patient apparently well. He may remain so for a considerable time,with intermissions of the symptoms of variable degree. The general condition of the patient is good,though slight anaemia may be present, accompanied by headache and dizziness. Thirst is common,but the appetite varies. Sometimes it is excessive,at other times diminished; possibly the patient is afraid to eat.

These symptoms may become chronic and give rise to special symptoms,or they may commence at the beginning.

The special symptoms referable to Gastric Ulcer are (1) Pain,(2) Vomiting,(3) Tenderness.

(1) Pain. (a) Its character. This is usually the most prominent feature of the symptom complex. It is described by the patients as burning,boring, gnawing or cutting. It is paroxysmal and occurs at the time of digestion. It radiates to the back between the shoulder blades,and is frequently relieved by food. Its position corresponds to the place that is most sensitive to pressure. If

the pain is continuous rather than paroxysmal, there is probably some complication, such as peritonitic adhesions to a neighbouring organ. This condition is usually brought on by alteration in the position of the patient.

(b) Its time of appearance. It occurs during digestion, either immediately after food or at the height of digestion. The time usually stated is half-an-hour to one hour, ~~or even later~~. Where the ulcer is situated near the pylorus, the pain occurs from 1 to 4 hours after food, and would in that case simulate a duodenal ulcer. Attacks of pain sometimes occur when the stomach is empty, e.g., at night, or even before meals. This is chiefly due to hypersecretion of hydrochloric acid irritating the ulcer. A small quantity of food relieves the pain for a time, but it re-appears later.

(c) Its localisation. Pain is felt in a circumscribed area in the epigastrium, usually to the left of the middle line, below the xiphoid, sometimes under the left costal edge. It is always constant in ~~in~~ its position. It frequently radiates backwards to the spine. Leube states that cicatrices rarely cause pain. Some authors have attempted to locate the position of the ulcer by noting the onset of pain after ingestion of food. This is of little importance, seeing that, as soon as peristalsis of the stomach wall occurs and the juice comes into contact with the ulcer, pain will be produced ir-

respective of the position of the ulcer.

(d)Its relation to food. The pain is aggravated by a large amount of food or solid food. Albuminous substances,such as egg and milk,give relief. The intensity of the pain varies according to the amount of gastric juice secreted. A non-irritating diet is the most suitable one,and if rectal alimentation is used the pain stops altogether.

In comparing the symptom of pain in my 50 cases with the foregoing,I find it was present in all of them,with the exception of one case,and this was one in which symptoms were latent. In dealing with its character,the patients have variously described it as being cutting,gnawing,boring,burning, and so on. The table below shews at a glance the character of the pain that was most frequent.

Character of Pain.

	Gnawing.	Boring.	Burning.	Stabbing.	Aching.
No.of Cases	15	9	15	2	1

	Cutting.	Shooting.	Pricking.	Dragging.	Cramp-like.	Agonising.
No.of Cases	2	1	1	1	1	1

The table shews that the commonest character of the pain was gnawing and burning.

Furthermore,it is of interest to note the frequency with which the pain appeared in paroxysms,its direction or radiation,and also whether it was present

continuously. The analysis is shewn in the subjoined table.

Pain occurring in paroxysms.	Continuous pain.	Radiation of pain.
40 cases	9 cases	27 to shoulder blades

The above shews that 40 cases had the pain in paroxysms and 9 had pain more or less continuous. The direction which the pain took was found to be towards the shoulder blades in 27 cases, and in 3 towards the heart. The remainder had the pain localised to the epigastrium.

Time of onset of the pain shews some variability, as the following table indicates. I have divided the periods of time and grouped them as follows. In each case the time denotes the first attack of pain after the taking of an ordinary meal.

Table to shew time of onset of pain after food.

3 minutes	5-10 mins.	10-15 mins.	15-20 mins.	20-30 mins.	½-1 hr.	1-2 hrs.	2-3 hrs.	4 hrs.
1	14	16	6	2	5	2	1	1

The lower figures denote the number of cases.

These shew that out of my 50 cases the most common period for the onset of pain after taking food was 10-15 minutes, and the next 5-10 minutes, and the third in frequency 15-20 minutes. The extremes are each represented by one case, one patient suffering pain within three minutes after food, and the other extreme shewing a case having pain as much as 4 hours after the taking of food. These figures contradict the time usually stated, namely, half-an-hour to one hour after food. It is a significant fact that three-fifths of my cases suffered acute agonising pain a quarter-of-

an-hour after taking food. It probably points to a very early and free secretion of the acid juice coming into contact with ulcers situated some distance from the pylorus. There was one case that had pain before as well as after food. This was probably due to an erratic secretion of the acid juice, similar to that in those cases where pain occurred at night. There were 11 cases out of the 50 that shewed pain during the night, chiefly about 3 or 4 o'clock in the morning.

It was found that pain was relieved in eight cases by taking food, such as milk, an egg or a biscuit. These patients even took food about with them to their work or ate it during the night. One patient used to squeeze his abdomen tightly so as to relieve the pain; two patients used to drink hot water, and one cold water. On the other hand, 20 patients stated that more food, no matter what it was, aggravated their pain; consequently there were associated with their pain the pangs of hunger. Two cases relieved their pain by alteration in their position. One of these two lay down in the recumbent posture, which relieved the pain instantly; the other was free so long as he did not make any violent movement, such as a sudden twist in bed.

The localisation of the pain was constant and definite, as the following analysis shews.

Localisation of the Pain.

In the Epigastrium.	Round the Umbilicus.	Diffuse.
43 cases.	3 cases.	3 cases.

The above shews that the most frequent site of pain was the Epigastrium, or "pit of the stomach," in the words of the patient. The area indicated was circumscribed and constant in position. In the majority of these instances the point was to the left of the middle line, lying under the left costal margin, usually one inch from the medial line. In some half-dozen cases it was lower, being about 2 to 2½ inches above the umbilicus and to the left. I had no case where it was situated to the right of the middle line. There were three cases in which the pain was located in the neighbourhood of the umbilicus (peri-umbilical), being ½-1 inch above, or to the left of it. Diffuse pain was experienced by three other patients. The pain was not localised, but was apparently felt all over the abdomen.

The duration of the pain was variable. The pain persisted and increased until vomiting or the taking of more food caused relief. The persistency of the pain in the series of 50 cases is shewn in the analysis below. This table gives an idea as to how long a patient can stand severe pain irrespective of treatment.

Duration or Existence of Pain.

	weeks			month	wks.	months					years
	1	2	3	1	6	2	3	4	6	9	1-2
Cases:	3	4	3	4	3	9	10	3	6	1	4

This analysis shews that 10 cases had gastric pain for

3 months,and 9 cases had gastric pain for 2 months. These patients had all complained of "dyspepsia"(i.e., sense of fulness and discomfort,flatulence and water-brash),and this had developed into the most distressing symptom of the symptom complex,namely pain. Only three cases had this severe symptom for a week,shewing the rapidity of the progress as compared with the four cases which experienced pain for 1 to 2 years.

(2) Vomiting.

Vomiting of food alone is fairly common,but not so constant as the pain. Out of 265 of Fenwick's cases, 72 had vomiting,while pain was present in 100%. Nausea and flatulence are more common. Where vomiting is present it usually occurs at the height ~~of the height~~ of the attack and relieves the excruciating pain. Some patients vomit only occasionally; others once a week; and yet others vomit after each meal,an hour or two after the pain has commenced.

The character of the food frequently determines the onset of vomiting. If the food be coarse and indigestible,vomiting frequently occurs after the excessive cardialgic pain. When vomiting does occur,the paroxysm of pain usually stops and thus relief is obtained.

The appearance of the vomit varies. If the food is completely digested,the material is fine. The digestion of albumins is good, on account of the action of the Hydrochloric Acid,which is increased. If the food is incompletely digested,the material contains

undigested particles. The odour is acid, and the material leaves an acid taste in the mouth. Chemically, the vomit contains a high percentage of acidity (due to excessive hydrochloric acid and not fermentative acids, e.g., acetic and lactic). Microscopically, the vomit shews well digested muscle fibres, but undigested starch grains. The vomit may contain blood, which need not necessarily arise from the ulcer.

The frequency with which vomiting occurred in my 50 cases is best shewn by reference to the table below, which also shews the nature of the vomit.

Number of Cases of Vomiting.	Nature of vomit.		
	Food.	Blood.	Food and Blood.
44	24 cases.	7 cases.	13 cases.

In 44 cases vomiting occurred. This number includes 24 cases in which the vomitus consisted of food only, 7 cases in which the vomitus was blood only - these were cases of haematemesis, and the vomiting occurred probably once or twice - and 13 cases in which food was vomited fairly regularly, to be followed by an attack of haematemesis, which secured them admission to hospital. Apart, then, from the 7 cases of haematemesis, vomiting of food occurred in 37 cases out of 50, or 70%, a figure higher than Fenwick's. Nausea was well marked in 35 cases; flatulence, heartburn and waterbrash in 37 cases. These symptoms were less complained of than usual.

Time of onset of Vomiting is seen in the analysis which follows. The periods are divided up into quarter-hours and denote the time of vomiting after taking food.

Time of Onset of Vomiting.

	¼ hour & under	½ hr.	¾ hr.	1 hr.	1¼ hrs.	2 hrs.	4 hrs.
Cases:	2	5	9	7	1	5	1

There were five cases where vomiting occurred unrelated to food. It will be seen that the majority of cases complained of vomiting within ¾ hour after taking food, there being as many as nine. At the end of one hour, seven cases are recorded as having vomited. These figures indicate times which are earlier than those recorded by Fenwick. There were five cases in which emesis occurred at the end of half-an-hour, and two at the end of a quarter-of-an-hour, or even before that. These numbers correspond to those in which rapid onset of pain was noted. Emesis occurred in five cases at the end of two hours, which apparently is more than usual. Speedy and rapid vomiting predominated in my series.

The five cases mentioned above were those of patients who vomited occasionally, the time being unrelated to any meal, for example, late in the evening, or before breakfast.

The frequency with which emesis took place is of interest and recorded below.

Frequency of Vomiting.

Occasionally.	2-6 times weekly.	3-4 times daily.	Twice daily.
7 cases.	14 cases.	7 cases.	8 cases

There were seven cases that vomited once only, and in that instance it was blood. Vomiting did not occur in six cases, or 12%. What is meant by "occasionally" referred to by the patient, is that emesis occurred

once or twice in a month or six weeks,or sometimes more often. The other periods are quite definite, and were given by the patients themselves. It will be noted that 2-6 times weekly is the most frequent period,as many as 14 cases coming under that heading. The frequency of twice daily is recorded in 8 cases, the emesis taking place in the middle of the day and in the evening. Vomiting after every meal,i.e., 3-4 times daily,occurred in 7 cases,or 14%; 16% vomited twice daily: 28% vomited 2-6 times weekly: 14% vomited occasionally: only 14% of cases did not vomit.

The vomiting,as stated,frequently occurs at the height of the excruciating pain,and in the majority of cases affords enormous relief. In the cases in which emesis occurred it was found that 25 got relief from vomiting. Some of these patients used to make themselves vomit by placing their fingers at the back of the pharynx. Relief was obtained occasionally by 8 patients,and not at all by two.

This table summarises these findings.

Relief was obtained in	Relief was not obtained in	Relief was occasionally obtained in
25 cases	2 cases	8 cases

In other words,50% obtained relief from pain; 16% of cases obtained relief occasionally; and 4% obtained no relief from pain,though vomiting took place.

(3) Tenderness. There are usually two painful pressure points that will be elicited on the examination of of a patient. One is epigastric, and the other dorsal. The epigastric pressure point is situated in the middle line, or to the left of it - seldom to the right. This same point is immediately below the xiphoid process. It is strictly located within a small area in the epigastrium. The dorsal pressure point is situated to the left of the middle line, in an area extending from the 7th. to the 12th. dorsal vertebra. Boas found this dorsal point in one-third of all cases of ulcer, and it is rarely found above.

There is also associated with these pressure points an area of hyperaesthesia of the skin, corresponding to the points of tenderness. As the healing of the ulcer occurs, the pain lessens, and concomitant with this there is diminution of the hyperaesthetic area and the points of painful pressure.

Tenderness was elicited in the abdomen in 48 cases out of my 50. This area was present in the epigastrium, lying under the left costal arch, below the xiphoid process, about one inch from the middle line, and was constant and circumscribed. In no case was it to the right of the middle line. Only in a few cases was it situated on the middle line. In every case it was located within a small area, confirming the usual condition of things. Pressure over this area invariably produced severe pain, frequently radiating to the back between the shoulders.

Associated with the anterior or epigastric pressure point, is a dorsal pressure point. Pressure on this point used to cause a return of the severe pain This dorsal tenderness or pressure point was present in 12 cases - about 24% - a figure much less than the one mentioned by Boas. In no instance was it found alone. The commonest position for this dorsal pressure point was to the left of and opposite to the spine of the 9th.dorsal vertebra. The next in frequency was the 10th.dorsal spine. The 7th.dorsal spine was noted in two cases.

Besides these tender pressure points, there is usually some hyperaesthesia of the skin, corresponding to the point of tenderness which may be the site of the ulcer. By drawing the finger nail gently over the skin in the epigastric area, there is evinced by the patient a sense of burning. This was found to be the case in 28 cases.

I summarise these observations in the table below:

Tenderness(Pressure Point).

Epigastric	Dorsal	Hyperaesthesia.
48 cases	12 cases	28 cases

This table points out that in 12 cases there were combined dorsal and epigastric tenderness, as well as hyperaesthesia of the skin of the abdomen. Furthermore, 16 cases exhibited hyperaesthesia of the skin as well as epigastric tenderness. Lastly, there were 20 cases in which epigastric tenderness was alone present. In no case was there dorsal tenderness or

hyperaesthesia of the skin present alone.

There are yet other symptoms associated with gastric ulcer,besides the three just enumerated. These are: (1) Haematemesis,(2) Melaena,(3) Acid Pyrosis, (4) Salivary Pyrosis, (5) Fever, (6) Gastro-intestinal Symptoms,(7) Genito-urinary symptoms, (8)Blood changes.

(1) Haematemesis. This symptoms does not always occur,though it is regarded as a cardinal sign. According to Brinton,it occurs in 28%,and to Gerhardt in 47%,whilst Lebert found it in 85%. It may be one of the first symptoms occurring in people in good health; but it more often occurs during the disease. The vomiting of blood can occur in erosions of the gastric mucosa,in tuberculous and carcinomatous ulcers, in cirrhosis of the liver and in circulatory disturbances of the stomach wall.

Haematemesis is favoured by physical exertion,emotional disturbances and menstruation. The amount of blood lost and the rapidity with which it is lost depend on the size of the eroded vessel. If severe,it leads to death,in which case the patient suddenly faints,turns pale,falls and dies within a few minutes. Death is not common as the immediate result of haematemesis. In this case one gets all the signs of an internal haemorrhage. The patient turns pale,does not become unconscious,hands and feet become ice-cold,pulse small and rapid and almost imperceptible. He frequently vomits,and the vomit consists of a quantity of dark

tarry fluid and partially coagulated blood. (HCl. acid has acted on the haemoglobin, converting it into haematin, and so the fluid is dark brown.) The stools the following day are also brown-black in colour. Sometimes the patient does not vomit, but the blood passes into the intestine. This occurs where the haemorrhage is slow and small in quantity. If the patient does not vomit the blood for some time, it will be found that when he does vomit the vomited material is coffee-ground in appearance, owing to the breaking-down of the red cells. The colour given to the vomit is formed by small brownish masses of blood pigment. The absolutely sure method of demonstrating the presence of blood in vomit is described by Weber,(22) who applied it to the spectroscope as follows.

Take a few drops of concentrated acetic acid and add it to the vomited matter previously diluted. Now shake up this mixture of acetic acid, water and stomach contents with one-third of the volume of ether. Let this mixture stand in the tube until the clear brown layer of ether separates on the top. If the separation of the ether is retarded, add a few drops of alcohol. This clear brown-red colour of ethereal extract is due to haematin. This body forms an acetic ether solution in sulphuric ether.

Nowplace a little of this brown-red ethereal extract in the spectroscope; four absorption bands are seen. Solutions of haematin in ether shew four absorption bands, and these are found

as follows:(1) one in the red; (2) one in the yellow; (3) one on the boundary between yellow and green; (4) one on the boundary between green and blue. The band in the red may be due to chlorophyll from the food; therefore dissolve the red blood pigment that is present in the acid ethereal extract,in alcoholic caustic potash. Then make the resulting fluid into a watery alkaline solution,so that it can be reduced by Ammonium Sulphide. Add Ammonium Sulphide to this alkaline watery solution; the fluid becomes red,and two bands appear in the green,which are characteristic of reduced haematin. Chlorophyll spectrum remains unchanged.

As regards my series,I find I had 28 examples ~~of~~ of this important sign of haematemesis out of my 50 cases,or 56%. This figure approaches Gerhardt's figure,which was 47%,though it exceeds it by 9%. Of these 28 cases,there were 6 in which there was a history of haematemesis during their period of indigestion; that is to say, the patient suffered for a considerable period with attacks of indigestion,and during one of these a haematemesis occurred. These periods varied from 2 months to 3 years when the haemorrhage took place. The subjoined table will make the above clearer.

Periods at which haematemesis occurred - divided into (a) recent and (b) old. By "recent" is meant the number of days or weeks before admission into hospital. By "old" is meant any period over two

months.

(a) Recent Haematemesis.

On day of admission.	On day of & 1 & 2 days before adm.	2 days before admiss.	On day of & 2 days before admiss.	3 days before admiss.
9 cases	1 case	4 cases	1 case	1 case

1 week before adm.	1 wk. and 1 day before adm.	6 days before admiss.	10 days before admiss.	14 days before admiss.	3 wks. before admiss.
1 case.	1 case.	1 case.	1 case.	1 case.	1 case.

(b) Old Haematemesis.

2 months before admiss.	6 months before admiss.	15 months before admiss.	2 years before admiss.	3 years before admiss.
1 case.	2 cases.	1 case.	1 case.	1 case.

The above table shews that 17 cases were admitted into hospital after a haematemesis occurring within one week of admission, nine of these having vomited blood on the day of admission. The other five cases vomited at varying periods within three weeks of admission.

It will be noted that 22 cases, or 44%, had this well-marked cardinal symptom of haematemesis, and it was this that brought them into hospital. This percentage, 44%, approaches Gerhardt's figure of 47%. The other percentage of 56 previously mentioned includes those cases in which there was an attack of haematemesis during their periods of illness going back for twelve years or so; that is to say, 12% of cases gave a history of having vomited blood at some period. These periods are tabulated above, and shew that one case vomited blood as long as three years ago.

One case of my series shews that haematemesis

was the first sign that anything of the nature of an ulcer was present in the stomach. His name was Reynaert,but his case will be referred to later.

It will be of interest to see at what period haematemesis occurred during an attack of dyspepsia. To do this I shall tabulate in two columns,one to shew duration of indigestion,the other to shew time of haematemesis.

Case.	Length of time of Indigestion.	Time of occurrence of Haematemesis.
E.A.G.	1½ years	3 months after onset of symptoms.
H.S.	16 years	16 years after onset of symptoms.
B.F.	1 year	10 months after onset of symptoms.
C.W.	2 years	18 months after onset of symptoms.
F.H.	6-7 years	3 weeks before admission into hospital.
E.C.	3½ years	6 months after onset of symptoms.
W.R.	2 years	10 days before admission into hospital.
J.T.S.	3 months	2½ months after onset of symptoms.
A.J.	6 months	6 days before admission.
F.S.	6 months	2 days before admission.
A.H.	10 weeks	day of admission to hospital.
J.D.	2 years	14 months after onset of symptoms.
C.H.	6 weeks.	5 weeks after onset of symptoms.
L.G.	3 months	3 days before admission to hospital.
F.J.	2 months	2 days before admission to hospital.
R.Y.	5 years	5 years after onset of symptoms.
J.Y.	2 years	2 years after onset of symptoms.
E.F.	2 years	2 years after onset of symptoms.
A.M.	2 years	2 years after onset of symptoms.
A.P.	2½ years	2½ years after onset of symptoms.
A.F.	2 years.	2 years after onset of symptoms.
G.G.	1 month	1 month after onset of symptoms.

N.P.	2 months	2 months after onset of symptoms.
S.H.	6 years	6 years after onset of symptoms.
J.G.	2 years	2 years after onset of symptoms.
E.B.	2 months	2 months after onset of symptoms.
N.S.	10 days	9 days after onset of symptoms.
F.B.	12 years	10 years after onset of symptoms.

From the foregoing it will be observed that considerable variance exists in the times of occurrence of haematemesis. The shortest time at which haematemesis occurred was nine days from the onset of symptoms. The longest time was 16 years after the onset of symptoms. Another extremely long time observed was ten years,and another was six years. Of course,these patients had intervals free from pain or other symptoms. The most frequent period at which the vomiting of blood occurred was two years after the onset of symptoms. The shorter periods of 1,2 and 3 months after the onset of symptoms were less frequent. Although haematemesis is a valuable sign,it does not become apparent for two years or so,as can be seen from my own statistics. Haematemesis occurred in the course of the disease,and in one case only were the symptoms latent. Furthermore,in nine cases the symptoms were recurrent,with the production of a haematemesis,after a lapse of some considerable interval without symptoms of any kind.

The quantity of blood vomited varied from a pint in seven cases to a basinful in two cases (measures more than a quart). Three cases are re-

ported as having vomited a quart each, and two cases a cupful. The cup referred to probably holds a little more than a gill. In three cases only did the amount of blood vomited prove fatal.

In the records mentioning what the patients were doing at the time the haematemesis occurred, I find that six were working at their usual occupations, two were walking, and three were lying in bed in hospital. The occupations of the six cases referred to were those of blacksmith, market porter, bottle labeller, tool maker and two domestic servants.

Relation of Haematemesis to the Sexes.

As was stated at the outset, the percentage of incidence of gastric ulcer is greater in the female than in the male. Hence it is natural to suppose that haematemesis, which is one of the most important cardinal signs, occurs more often in the female than in the male. This can be seen by reference to the following table, which shews the number of males and females who had haematemesis as a symptom.

Female	Male
20 cases	8 cases

It will be remembered that among my 50 cases there were 33 females and 17 males; and so it will be seen that 20 out of 33 females and 8 out of 17 males, or 60% and 48% respectively, suffer from haematemesis. These figures are high, and serve

to shew the value of haematemesis as a sign. This is the most frequent cause of the admission of patients into hospital. Haematemesis occurred in six cases after admission to hospital, in three of which it was fatal. One case had three attacks of vomiting, and another had two.

(2) Melaena, or Occult Bleeding.

This, of course, may follow haematemesis, though it can occur apart from it. The blood is black in colour and of tarry consistency, owing to the change of haemoglobin to haematin and the formation of iron sulphide by the action of sulphuretted hydrogen in the bowel. The patient may not notice that his stools are black, but may complain of feeling faint after an evacuation, or his appearance may suggest internal haemorrhage.

Occult bleeding occurs in cases of haemophilia, purpura, scurvy, ankylostomiasis, typhoid fever, arteriosclerosis, malignant tumours of the intestinal tract, haemorrhagic pancreatitis, tuberculous and syphilitic ulceration of the bowel, haemorrhoids, fissures and fistula. When examining faeces for occult blood, it must be remembered that raw or badly cooked beef, sausage, and articles of diet containing blood will give positive reactions. Hence some preliminary dieting is necessary before examining for blood. It should be stated that well boiled or roasted meat does not interfere with the test.

I had an opportunity of observing 21 cases

which exhibited blood in the stools. Of these there were 14 in which haematemesis had occurred either on the day of, or one or two days before, admission into hospital. Consequently the existence of blood could be ascertained by naked-eye inspection. The stools were typical - dark brown or tarry black in colour, with a consistency that was solid and somewhat constipated. No recent red blood was observed in the stools.

To demonstrate to the students at the General Hospital, Birmingham, the tests mentioned below were carried out.

The other 7 cases were patients who had haematemesis one to two weeks before admission; so it was in this type of case that careful testing was necessary.

In all cases was it observed that meat, sausage or blood-containing articles of diet were eliminated 48 hours before the collection and testing of the faeces. In each of these 21 cases, I was able to prove by any of the methods mentioned later the presence of blood in the stools. In the recent haemorrhages the spectroscope gave evidence of blood pigment. The spectroscope was not invariably used, unless for purposes of demonstration. The production of Teichmann's crystals was a constant feature in well-marked cases of haematemesis. The chemical tests, especially the Benzidin , gave well-pronounced results, even in some of the long standing cases. The blood could be found in the

stools for as long as a fortnight after the occurrence of a severe haematemesis. In the three cases which were fatal,altered blood was present constantly in the stools.

The following tests were employed by me and performed as described. These were all demonstrated to the students who attended the hospital. (1)Microscopic for blood cells and pigment. (2) Spectroscopic. (3) Teichmann's crystals. (4) Chemical.

(2) In the spectroscopic test,dissolve a portion of the faeces in a small amount of water; add concentrated acetic acid: filter: shake up filtrate with ether. If blood is present,ether becomes reddish-brown and the spectrum shews the broad absorption bands of acid haematin in the red.

(3) To produce Teichmann's crystals - mix the faeces with sodium chloride crystals on a glass slide and cover with a cover-slip. Allow glacial acetic acid to trickle beneath the slip: now heat the slide over a flame: add glacial acetic acid drop by drop,until the fluid becomes brown,and allow to cool. Under the microscope ~~the microscope~~, red-brown rhombic plates of haematin hydrochlorate (haemin) are seen.

(4) Chemical Tests. These are many,but the Guaiac Resin and Benzidin tests are the most reliable and most often performed. If the faecal matter is hard, break it up and soften with as little water as possible; add a small quantity of glacial acetic acid and thoroughly mix. To the mixture now add 30 c.c.

of ether and let it remain for several minutes, thoroughly shaking the while. To a portion of the extract, which is clear, add an equal quantity of distilled water, and shake. Now add to this mixture a few granules of powdered guaiac resin, and allow it to dissolve. To this solution add 30 drops of chemically pure turpentine and mix well. If blood is present, a distinct light blue colour appears, but disappears after a short time.

The test I most frequently performed was the Benzidin test, which is the one most often applied and first demonstrated by Adlers in 1904. This may be carried out as a "wet" preparation or with specially prepared Benzidin paper. The "wet" preparation consists of the following. Mix well 2 c.c. of water with some faeces(size of a bee) and heat to boiling point. The reagent to be added now consists of 10 or 15 drops of benzide solution(prepared by placing a small quantity of benzidin in 2 c.c. of glacial acetic acid and shaking quickly) and 2 or 3 c.c.of a 3% solution of hydrogen peroxide. Then mix. If blood is present, a green or blue colour appears on the addition of the reagent to the solution of faecal matter. This test was successful with all my cases. Benzidin paper reaction was devised by Einhorn as a simpler method. The paper is made by moistening filter paper with a saturated solution of benzidin and glacial acetic acid and drying. A piece of benzidin paper is immersed in the solution to be

examined and a few drops of hydrogen peroxide added. A blue colour appears within a minute, if the reaction is positive. If benzidin paper is used for gastric haemorrhage,it must be remembered that hydrochloric acid itself develops a blue colour beyond two minutes. Likewise farina and boiled potato produce a positive reaction; so the liquid portion of the gastric contents is alone used. The patient in this instance must be dieted on a lacto-farinaceous diet,as salts of iron and saliva,sweat and chlorophyll(from vegetables) all give positive reactions.

The value of determining the presence of occult blood is borne out by the fact that it is a premonitory sign of some lesion in the alimentary tract high up,acts as a differential diagnostic sign,and helps to diagnose between carcinoma and ulcer especially. In the former, melaena is constantly present; whilst in the latter it is intermittent. It has a value for diagnosis,prognosis and therapeutics.

(3) Acid Pyrosis(waterbrash). This is quite a common symptom and is due to the excessive secretion of hydrochloric acid,which is regurgitated into the mouth. At the same time there is a burning sensation,either in the praecordium or in the sternal region,and it has been termed "heartburn." If fermentation is present from the retention of the stomach contents in a dilated organ,the acids produced are lactic,butyric and

acetic. These cause symptoms of "heartburn." The production of this condition has been attributed by some to relaxation of the cardiac orifice associated with excessive peristalsis of the stomach.

(4) Salivary Pyrosis. Profuse salivation is often associated with gastric ulcer. The saliva is secreted in huge quantities,coming in gushes,and then swallowed. It helps to neutralise the acidity,and possesses diastasic properties. These attacks may be occasional or occur as often as once a day.

(5)Fever. This is usually absent,unless there are complications,which are usually haemorrhage or peritonitis. If the temperature rises suddenly and the patient collapses at the same time,there is haemorrhage occurring. The temperature soon falls, however. In my series there were 13 cases in which pyrexia was present. One case developed a temperature of 101°F.,with a pulse of 100,after an operation for gastric ulcer. This pyrexia lasted three days and was associated with bronchial catarrh. This,of course,was an accidental occurrence,and unrelated to the gastric ulcer. Another case developed a temperature of 99°F.,with a pulse of 90,for three days prior to operation,after which the temperature and pulse returned to normal. A case on admission after a haematemesis(one day before admission) had a temperature of 100°F. with a pulse of 96,which remained for two days and then fell to 98°F.and 84. A fourth case was brought in collapsed after a severe haematemesis and registered a

temperature of 96.8° F. and a pulse of 84 to the minute. After 24 hours there was a slight rise of temperature to 98°F. and a further rise to 99°F.; it regained the normal height at the end of another 24 hours. A similar case to the one just mentioned had a temperature of 97°F. and a pulse of 80 to the minute on admission. This was also after a haematemesis. At the end of 48 hours, the temperature and pulse returned to normal, and they remained so during his stay in hospital. Another example of a haematemesis occurred in a female, who on admission had a temperature of 99°F. with a pulse of 84 to the minute. These soon returned to normal, however, but on the seventh day after admission she developed tonsillitis, which raised the temperature to 103°F. and the pulse to 98. These were raised for three days only, after which both resumed the normal. Another patient had a temperature that registered on admission 100.4° F. and a pulse of 100 to the minute. For the first week the temperature and pulse varied between 100°F. and 101°F. and 100 and 120 per minute, respectively. They then returned to the normal. No cause was assigned for this slight pyrexia. Similarly, another patient remained without signs of fever for three weeks, but during the fourth week developed a temperature of 101°F., though the pulse was little elevated. The temperature fell to normal in three days. The following is another illustration of rise of temperature occurring as the result of more than one haematemesis, the rise being coincident

with the death of the patient. On the day of admission, after a severe haematemesis, the temperature was 97°5 F. and the pulse 84 per minute. Four days later the temperature rose to 100° F. and the pulse accelerated to 112, at the same time as another attack of haematemesis, which caused his death. A similar example was observed in another patient, who, on admission, had a temperature of 98°.4 F. and a pulse of 84. Four days later a haematemesis occurred, the pulse was quickened to 96, but the temperature remained the same (98° F.). Two days after this another haematemesis occurred; the pulse was rapid and amounted to 120 per minute, the temperature having fallen to 97° F. The patient was then operated upon, but the temperature now rose to 102° F. and the pulse accelerated to 130 per minute. At this juncture a third haematemesis occurred, which caused the patient's death. Another similar instance was in a female patient, who on admission had a pulse of 92 per minute and a normal temperature. Three days later the pulse increased to 108 per minute and the temperature registered 101° F.. After three more days, both pulse and temperature returned to normal, and remained so for a week. At the end of that time a fatal haematemesis took place; the temperature just before death registered 95 F. and the pulse 100. My other cases agree with those usually described as running a normal course, unless a complication occurs, in which case an alteration in the temperature

and pulse is observed.

(6) Gastro-intestinal Symptoms.

The tongue varies: frequently it is red, clean and moist; if anaemia be present,it is pale and flabby; in the more chronic cases,it is covered with fur. In all my cases,the tongue was coated with brownish or white fur and the breath at the same time had a disagreeable odour. The teeth with few exceptions were bad,and in 10 cases were removed. These patients improved considerably after the removal of the bad teeth,as it lessened the pyorrhoea alveolaris.

The appetite is sometimes excessive,but the patient is usually afraid to eat. This was so in the majority of my cases,for as soon as the patient had got rid of the pain he invariably ate well. Thirst is usually well marked,and the difficulty is to allay it,especially when a patient is on measured quantities of fluid. Constipation is usually present,and was so in 24 cases. Diarrhoea is rare. I had no case where diarrhoea was present.

(7)Genito-urinary Symptoms.

The urine varies; sometimes it is normal; at other times it contains albumoses,albumin,acetone & diacetic acid. Chlorides are frequently diminished whereas the phosphates are increased. Indican is present if there is fermentation in the stomach. The presence of acetonuria and diaceturia is now well established,and these bodies of themselves

give rise to a train of symptoms which resemble those due to an acid intoxication. When the symptoms are present in a mild form, the patient complains of epigastric pain, persistent vomiting, headache and giddiness. In the more severe forms there are drowsiness, dyspnoea and coma. The acetone is recognised from the smell of the breath and urine, as well as from the iodoform and nitro-prusside tests. Acetone and diacetic acid are found in other gastric disorders besides ulcer, chiefly dilatation of the stomach, chronic gastric catarrh and enteritis.

I had in my series 14 cases whose urine was not normal. There were present phosphates, glucose, indican, albumin and acetone and diacetic acid, as the following table shews.

Contents of Urine.	Number of Cases.
Albumin	1 case
Phosphates	10 cases
Indican	1 case
Acetone and diacetic acid	1 case
Glucose	1 case

This analysis shews that one case had albuminuria; it was not large in amount - chiefly a faint haze with nitric or picric acids. This cleared up before the patient left hospital. Phosphates were present in the urines of 10 patients. Of these, there were four cases associated with some degree of dilatation of the stomach. In one case, indican was found in the urine. There was some enlargement

of the gastric organ in this instance. Acetone was combined with diacetic acid in the urine of one patient(a female). This patient died from a haematemesis. In this case the patient's pain persisted as well as the vomiting and epigastric tenderness. One patient(a male) suffered from diabetes (glycosuria) and developed a gastric ulcer. Fourteen days before admission he had a severe haematemesis. The degree of glycosuria was not great; the usual quantity of sugar passed in the 24 hours was 572 grains. It should be mentioned that this patient also had arteriosclerosis. There was no diacetic acid or acetone in the urine.

(8)The Blood Count usually shews a chlorotic character. The red cells are diminished,being reduced to about 4,000,000 per c.m.m. Haemoglobin percentage is about 58% and index about 0.72. The white cells number about 7,500 per c.m.m.: differential count shews a slight relative increase in the small mononuclear cells. In my own cases the red cells varied in number from 2,300,000 per c.m.m.to 4,500,000 per c.m.m. The haemoglobin percentage ranged from 45% to 60%,and the index from 0.65 to 0.75. The white cells were in number about 7,200 to 7,500 per c.m.m.,except in one case,which was one of a subphrenic abscess, where the white count was 15,000 per c.m.m. The blood pressure variously ranged between 150 m.m of mercury to 115 m.m.

The general nutrition of the patients remained good for some time,depending often enough on the duration of the disease. In those cases where the nutrition was impaired,the majority of the patients presented the following appearance: pallid,thin face, with a somewhat anxious expression; sunken eyes; flabby muscles and general loss of subcutaneous tissue,and varying degrees of anaemia.

Objective or Physical Signs.

There is little positive evidence to be gained from an abdominal examination in an uncomplicated case. The region of the stomach does not protrude,unless there is gastrectasis; not infrequently it is sunken.

Palpation results in the finding of points of tenderness. A point is most often found just below the xiphoid process in front,and another point,less frequently present,to the left of the 11th.or 12th. spinal vertebra. These two points are from about 2 to 3 c.m. in diameter,and pressure on the anterior point frequently causes the pain to shoot back to the posterior one. Diffuse soreness may be complained of in the epigastrium instead. Stroking the finger downwards over the lower costal margin towards the umbilicus causes a burning sensation in the same nervous segment that supplies the stomach. This same movement produces usually an exaggerated skin reflex in that area.

Where there are adhesions of the stomach to

the neighbouring viscera,a distinct lump is frequently felt in the epigastrium. Chronic and thickened ulcers are sometimes palpable.

From the analysis of my notes,I find that in uncomplicated cases,of which there were twelve, the abdomen presented no unusual features to the eye. No abnormal flattenings or bulgings were found. Respiratory abdominal movement was unrestrained,and the movements of the abdominal muscles caused only slight discomfort. In five of these cases the abdomen was somewhat sunken,shewing a depression in the epigastrium,the costal margins appearing prominent. Palpation in each instance produced tenderness over a circumscribed area in the epigastrium. This point has been mentioned previously as lying below the xiphoid process,about one inch from its tip and one inch from the middle line. This point of tenderness frequently corresponded to the area where the gnawing pain was felt worst. In one case the pressure point of tenderness lay on a line in its inner third,between the umbilicus and anterior superior iliac spine. In yet one other instance the point of tenderness differed in lying under the left costal arch,at the junction of the 8th.rib and the 7th.. The point of tenderness thus described is the anterior or epigastric,and pressure on it causes the pain to radiate to the back between the shoulders in most cases. Sometimes radiation is towards the praecordia,or downwards to the flank.

There is another point of tenderness, which is situated dorsally, as described before. Four of the twelve cases shewed this. The area of tenderness is cirucmscribed, and is about the same size as the epigastric. This point lies opposite the 9th. dorsal vertebra.

Palpation elicited one other fact, namely, resistance of the abdominal muscles to the pressure of the hand. Abdominal rigidity was observed to some extent in all the uncomplicated cases. The rigidity is confined to the upper belly of the left rectus muscle. This belly is resistant to fairly deep pressure.

Hyperaesthesia of the skin was found in five of the twelve uncomplicated cases. This was demonstrated by stroking the skin with the finger from the left costal arch downwards towards the umbilicus. This movement, when sharply done, produced an exaggerated and rapid reflex contraction of the muscles on the left side.

There were 38 complicated cases, of which there were 9 cases of recent haematemesis (that is, on day of admission), 6 cases of old haematemesis (that is three months before, and over), 7 cases of dilatation of the stomach, and two cases of subphrenic abscess. Inspection of the abdomen revealed in a case of recent haematemesis that it was sunken in the upper portion, above the umbilicus, and that it moved with

respiration. Palpation revealed marked resistance of both upper bellies of the recti muscles. Tenderness was elicited in the epigastrium, below the xiphoid and to the left of it. Liver and splenic dulness were unaltered. The signs were those of an acute abdominal trouble localised to its upper portion.

In the cases of old haematemesis, the abdomen was apparently quite healthy. The subcutaneous tissue was deficient, and thus the bones of the pelvis and the ribs stood out prominently. The upper portion of the abdomen was scaphoid, and pulsation of the aorta was more noticeable. Otherwise, there was nothing to shew that a haematemesis had occurred at any time. Palpation was likewise negative, slight resistance and tenderness being present in some cases.

In cases of dilatation of the stomach, a fulness was to be seen occupying the upper half of the abdomen. Sometimes peristalsis could be observed, passing from left to right. Percussion revealed a tympanitic note, and, with palpation, splashing was heard.

The physical signs of subphrenic abscess will be discussed under that heading.

The inference to be drawn is that the physical signs of the abdomen in a case of gastric ulcer are negative, unless some complication, such as perforation, is present.

Clinical Forms of Ulcer.

Owing to the changeable character of the clinical picture of gastric ulcer, there must be, of necessity, innumerable varieties of ulcer. Out of the maëlstrom of description of these numerous ulcers, the following regular types may be distinguished.

(a) Latent. In this form, at its commencement, there are no symptoms, the patient usually complaining of nothing, or sometimes of a slight discomfort after food or even a little flatulence. There are no symptoms to warrant the diagnosis of an ulcer, or to give a clue as to the dangerous condition, until suddenly a violent haematemesis or perforation occurs. As an instance, I quote the case of William Reynaert, aged 26 years, a policeman, who was quite well until ten days before admission into the General Hospital, Birmingham, on April 27th., 1910. He stated that he felt "low" and depressed, had no appetite, and had a feeling of giddiness. He experienced some nausea, but no actual vomiting. He further complained of restlessness and spots in front of his eyes. He had some slight discomfort after food for three or four days, but no actual pain. Then suddenly at night he vomited a quart of blood, which was dark-coloured, and soon after he was brought to hospital. In this case, there was no suggestion of the presence of a gastric ulcer. This patient died on May 9th., ten days after admission.

(b) Acute Haemorrhagic Ulcer. This,as just stated,commences without any symptoms. Suddenly a haematemesis,often fatal,occurs. If the patient does not succumb to the profuse loss of blood,signs of severe secondary anaemia develop. This development of secondary anaemia seems to act as a safeguard to the patient,as the symptoms gradually subside and the patient improves - cf. Reynaert. I had seven cases of this type of ulcer, which I give in detail.

Case I. W.Reynaert,previously mentioned as having latent symptoms,was admitted in a condition of great pallor,and with all the appearances of having lost a great quantity of blood. The lips and mouth were parched; the tongue was covered with a brown-red coating; the patient's limbs were cold,and he was extremely nervous. The pulse was small and wiry, numbering 84 to the minute; the temperature was 98. The abdomen moved freely on respiration. He complained of no pain,but some slight diffuse tenderness over the epigastrium was elicited on gentle palpation. Melaena was present. His progress was not satisfactory,as he complained now of occasional pain in the abdomen. The signs of the recent loss of blood were still well marked,as well as melaena. On the fifth day after admission he assumed an anxious expression and became very restless,and his extremities got very cold. There were two attacks

of faintness on that day,followed by increase of blood in the stools. His pulse was 96 per minute, regular but jerky; and the blood count shewed considerable anaemia,the red cells numbering 2,300,000 per c.m.m. The haemoglobin percentage was 45. On the following day the pallor of his body increased. No vomiting occurred at all. The temperature on this day was 99.4 F.; the pulse was 96,regular, fairly good volume,and jerky. Suddenly another change occurred,his temperature falling to 97 F. and the pulse increasing to 120. His restlessness increased. It was decided to operate,and it was found that the stomach was slightly dilated. Recent adhesions between the posterior surface of the stomach and the stomach bed(across lesser peritoneal sac)were seen. Two ulcers on the posterior aspect were seen near the lesser curvature. Posterior gastroenterostomy was performed. His progress after the operation was unsatisfactory,the pulse registering 130 per minute and being weak. Blood pressure was 115 m.m. of mercury. No pain or vomiting. Skin remained wax-like. Tinct.opii ℥ iv,6-hourly,by the mouth, were given for restlessness. On the day of death,May 9th.,pulse increased to 160 per minute and temperature rose to 102 F. The post-mortem revealed one small punched-out ulcer on the posterior aspect,near the lesser curvature,half-way between the cardiac and pyloric orifices. There was no thickening around the ulcer. At the base of

the ulcer,two small orifices of arteries were visible. Close to the above ulcer was a smaller one,but very superficial. A small area of puckering was seen in the same region - the sign of an old healed ulcer.

Case II. Florrie Jones,aged 35,a domestic servant, was admitted into the General Hospital,Birmingham, on Jan.15th.1911,after having vomited a large quantity of blood. She had attended the out-patient department,two months previous to admission,for gastric ulcer. Two days before admission she vomited over a pint of blood. Her condition was that of a pale woman,fairly well nourished; the tongue was furred and the breath offensive. The abdomen was tender all over,especially so under the left ribs. The upper bellies of the recti muscles were rigid. Melaena was well marked. Her progress was slow; the epigastric tenderness increased,especially in the region of the spleen,the pulse remained at 104 to the minute,and the temperature at 100 F.. On the fourth day after admission it was decided to operate. There had been no vomiting,but pain was considerable. Mr.Barling operated on Jan.19th.,and found a large ulcer on the lesser curvature,near the pylorus. Posterior gastroenterostomy was done. Unfortunately the patient did not improve,as she still complained of pains in the region of the stomach. On Feb.1st.it was decided to start rectal feeding,nutrients alternating with saline solution every six

hours. She seemed to improve somewhat up till Feb.9th.,when she suddenly collapsed and died. At the autopsy it was seen that a large ulcer was situated on the lesser curvature,near to,but not quite at,the pylorus. It extended on to the upper part of the anterior wall,and to a greater extent on to the posterior. The edges were clearly cut,as though punched out. The wall of the stomach was in part eroded completely,and the viscus was adherent to the diaphragm as well as to the under surface of the liver. Elsewhere in the floor,which was still formed by gastric coats, eroded blood-vessels were seen. The gastroenterostomy junction was quite healthy and shewed no signs of leakage. Peritoneum shewed general suppurative peritonitis,probably secondary to the ulcer and not to the operation,as all surgical wounds were sound.

Case III. Charlotte Hardwick,aged 40,doing house-work,was admitted into the General Hospital, Birmingham,on Oct.12th.,1910,with the complaint of pain and vomiting after food for six weeks. Previous to admission,she had been in bed 14 days. For a month prior to entrance into hospital, she had had great discomfort,flatulence and distension of the stomach,with severe pain,four hours after food. Vomiting always relieved her. The pain increased in severity,especially after taking food. She noticed that the stools began to get black three

weeks before admission,and a fortnight later she vomited a cupful of blood. The woman on admission was thin and pale; the mucous membranes were anaemic; the pulse was small and counted 110 per minute; the blood pressure was low; and the temperature was 98.8 F.. Systolic murmurs were heard at the mitral and tricuspid valves. Epigastric tenderness,with rigidity of the rectus muscle,were elicited on gentle palpation. Indican and sugar were present in the urine. The patient's condition did not improve,as the abdominal tenderness increased,vomiting persisted,pain became burning and flatulence was frequent. On Oct.28th., 16 days after admission,the patient suddenly vomited a very large quantity of blood and then collapsed and died. The autopsy shewed that the stomach was filled with blood-stained fluid,and on the posterior wall there was a round ulcer, with sharply-cut edges, penetrating to the peritoneal coat,which was injected and slightly puckered. The mouth of a vessel of some size stood out prominently.

Case IV. Nora Scorfield,aged 15,a domestic servant, was admitted into the General Hospital,Birmingham, complaining of severe pain in the abdomen and frequent vomiting of food and blood for ten days. She had been complaining only for 14 days of severe cutting pain in the middle of the back and stomach, commencing half-an-hour after food and lasting half-an-hour,when it was relieved by vomiting. Two days before admission she suddenly vomited blood. Me-

laena was present. Her condition on examination was that of a pale and anaemic girl; the tongue was furred; there were haemic murmurs at all the orifices of the heart. The abdomen was concave, and palpation elicited tenderness just below the xiphoid cartilage and some rigidity in the upper bellies of the recti abdominis. Dorsal tenderness was also present. Pulse was of good volume and strength and counted 98 per minute; temperature 98 F. This patient made an uneventful recovery.

Case V. Gertrude Griffin, aged 25, occupation not stated, was admitted into hospital, July 25th., complaining of severe pain five minutes after food, especially after cold food. There was no vomiting, except a sudden haematemesis on day of admission. Severe heartburn and waterbrash were present. She had had these symptoms for one month previous to admission, and her condition then was that of a girl with a pallid face and mucous membranes very anaemic. The abdomen was sunken, and there was tenderness in the middle line, 1½ inches below the xiphoid cartilage. The tongue was furred, and the breath had a foetor. Dorsal tenderness was present also. This patient improved well and was discharged cured.

Case VI. Eleanor Bott, aged 17, a bottle labeller, was admitted into the General Hospital, Birmingham, on Aug. 29th., having vomited blood on the day before as well as on the day of admission. She had for two months been complaining of slight dyspeptic

symptoms, such as nausea, giddiness and tinnitus aurium. Pain occurred ten minutes after food, but there was no vomiting. She had two attacks of faintness, and a sinking feeling in the abdomen, one week before admission. On examination, the girl was pale, with anaemic mucous membranes, and lay quietly in bed. There was tenderness under the xiphoid process, with resistance of the muscles on the left side of the upper portion of the abdomen, which moved freely on respiration. The pulse was dicrotic and regular. There was a well-marked systolic murmur at the mitral orifice, conducted into the axilla. A similar murmur was heard at the pulmonary orifice, the second sound being reduplicated in the same area. Melaena continued for ten days after admission. The progress of the patient was very satisfactory, and she was discharged cured.

Case VII. Arthur Horton, aged 39, a market porter, was admitted into the General Hospital, Birmingham, on July 22nd., suffering from profuse haematemesis. He had had abdominal pain, which commenced ten weeks prior to admission and gradually got better. On the day of admission he suddenly felt faint and immediately vomited blood, about a pint. His condition was serious, as he was so pallid. The tongue was coated with fur, and his mouth had an offensive odour. The abdomen was sunken and moved with respiration. There was some tenderness below and to the left of the xiphoid process. Resistance to

the palpating hand was encountered over the upper portions of both recti. The pulse was feeble and at the rate of 84 per minute. The temperature registered 97.5 F.. Progress was not good,as on the third day after admission he had another severe attack of haematemesis. Melaena was well marked. Segments of taenia mediocanellata were evacuated along with the motions; the head of the tape-worm could not be found. On the fourth day another profuse haematemesis occurred,which increased the pallor of the patient and increased the pulse rate to 112 per minute. The temperature at the same time registered 100 F.. More segments of the worm were passed. Still later on the fourth day,July 26th.,a last and fatal haematemesis occurred. The post-mortem examination showed the stomach to be full of blood,and after it was cleared away,a large and deep ulcer,with well-cut edges,was observed on the anterior wall. The ulcer was a square inch in size,and had penetrated through all the coats of the stomach to the peritoneum,which was glistening and smooth,not thickened,but injected. In the base of the ulcer was the opening of a large artery. The jejunum contained two intertwined specimens of taenia mediocanellata.

Out of seven cases,four were fatal;which indicated the danger of an ulcer of this type.

(c) Recurrent Form. In this variety the ulcer runs a progressive chronic course. There are frequent attacks of vomiting of blood, though the loss of blood is inconsiderable. The stools in these cases are almost always black and contain blood pigment. These patients get very much reduced in weight, their condition is poor, and they often develop a cachectic condition. I found that the greater number of cases in my series belonged to this group. There were 18 cases. I shall give a brief outline of each to bring out the important points.

Case I. Norah Pearson, aged 26, lathe worker, was admitted into the General Hospital, Birmingham, on Sept. 14th., with profuse haematemesis. She had had pain after food on and off for six years, with occasional vomiting. The pain and vomiting became aggravated two months before admission, and later the pain was almost continuous. Faintness overcame her ten days before she came into hospital, and on the day of admission she vomited about a pint of blood. She was a pallid, anaemic and somewhat badly nourished woman. The tongue was furred, the teeth were bad, and the breath had a foetor. There was marked tenderness in the middle line, just below the xiphoid cartilage. Melaena was present, and had been for a few days before admission. There must have been a slight haemorrhage the day she fainted.

Case II. Elizabeth Caldicott, aged 20, worker in a warehouse, was admitted into the General Hospital, Birmingham, on Jan.6th., 1911, from the out-patient department. She had had pain for three years, starting in 1908; she vomited a large quantity of blood in November of that year. As she did not improve, Mr.Heaton operated on Jan.28th., 1909, and performed a posterior gastroenterostomy. (The surgical notes made no statement as to whether an ulcer was found or not.) Soon after being discharged, this patient began vomiting food several times daily; later she vomited only occasionally. The vomit was sometimes tinged with blood. She noticed that her stools became darker at different periods. The pain now on admission was knife-like and short between the shoulders, appearing 10 to 15 minutes after food. She was pale and anaemic-looking, and not well nourished. The abdomen shewed the scar of an old operation wound. It was soft, and pressure in the epigastrium caused some pain. She vomited liquid material several times for four days after admission. This condition may have been due to symptoms of the old ulcer, which had not healed after the operation, or to the fresh formation of a peptic ulcer in the jejunum.

Case III. Fred Hopkins, aged 39, occupation not stated, was admitted to the General Hospital, Birmingham, on May 1st., complaining of a hungry, gnawing pain three hours after food, with flatulence and vomiting several times a week. The

pain had been present six or seven years,with intervals of freedom from attacks. Three weeks before admission,he was suddenly seized with fainting,fell to the ground,and vomited a large quantity of blood. Whilst in hospital,he made slight progress,and,a favourable opportunity occurring,it was decided to operate. Mr.Lucas, on May 22nd.,found a very large ulcer,extending along the lesser curvature into the lumen of the stomach; it was impossible to excise it,but a posterior gastroenterostomy was performed.

Case IV. Louise Hemms,aged 43,doing house-work, was admitted into the General Hospital,Birmingham,on June 30th.,1911,from the out-patient department. Her previous history was that on Nov.21st.,1908,Mr.Barling operated on her for gastric ulcer and found two ulcers in the stomach,and ever since she had complained of a pricking sensation,accompanied by waterbrash. She became worse 14 days before admission,having severe epigastric pain about 1-2 hours after food, which she vomited for the first time a week before coming into hospital. The vomit was yellow, streaked with blood,and smelt acid. On examination,the old operation scar was visible,and there was some tenderness immediately under the ensiform cartilage,with resistance of the left side in the upper part of the abdomen. As in Case II, this might possibly be due to the formation of a peptic ulcer in the jejunum,or to a recrudescence

of the old ulcer.

Case V. Fanny Brown, aged 39, tailoress, was admitted on July 23rd. into the General Hospital, Birmingham, from the out-patient department. Her previous history shewed that twelve years before she had had severe pain and vomiting after all food. This got better under a three-months' course of treatment. A relapse occurred two years before admission, after a latent period of ten years, and this time the attack was associated with haematemesis. She made a good recovery. Then two months before admission pain and vomiting returned. The pain occurred ten minutes after food, and persisted until vomiting relieved it, a quarter-of-an-hour later. There were waterbrash, heartburn, flatulence and burning in the throat. Her teeth were bad, and her breath had a bad odour. The tongue was furred. There was some pain and tenderness over the epigastrium, and the abdomen moved somewhat restrainedly.

Case VI. Elizabeth A. Good, aged 45, doing housework, was admitted into the General Hospital, Birmingham, on Jan. 11th., with severe pain between the shoulders and "round the heart." Previous history shewed that fifteen months before she had had a haematemesis, from which she made an indifferent recovery, as the pain had been present for the last year, commencing ten minutes after meals. She had not vomited lately. She was a pale woman, poorly nourished, and the tongue was dirty. Epigastric

pain and dorsal tenderness were present,with resistance in the upper abdomen.

Case VII. Beatrice Figgures,aged 22,domestic servant,was admitted from the out-patient department on Jan.12th.into the General Hospital, Birmingham,with the complaint of pain in the chest. Previous history shewed that twelve months before she began to have a gnawing pain and vomiting after most meals; the appetite was good,but she was afraid to eat. She was treated for six weeks without benefit,as she was still at work; so a month's rest was taken,which cured her. Two months before admission,she vomited 1½ pints of blood,as she was walking in the street; half-an-hour later she vomited some more blood. The pain got increasingly worse,and became constant a fortnight before admission. The abdomen was soft on palpation,but some slight epigastric tenderness was present. There was no rigidity of the abdominal muscles.

Case VIII. James Dowling,aged 33,a bricklayer's labourer,was admitted into the General Hospital, Birmingham,on June 24th.,with abdominal tenderness and pain. There was a rapid pulse and some rigidity of the upper part of the abdomen. Previous history shewed that he had been suffering from indigestion for two years. It started with pain in "the pit of the stomach" immediately after food. There was flatulence,but no vomiting. He always woke up with a "bad" taste in the mouth. Six months ago he vomited blood for the first time(a basinful); after

this he persistently vomited food for three weeks. This stopped,however,under medical treatment. The pain got increasingly worse,until it became continuous,and vomiting restarted and occurred as often as three or four times a day. He vomited blood again one month before admission. His stools were always dark. On examination,he was found to be a thin,pale-faced man who had lost some weight. He lay on his back. The tongue was furred; there was no rise of temperature; the abdomen was painful and tender in the epigastrium,the liver a little big. Whilst in hospital,he vomited,and the pain and tenderness increased; so on July 3rd.he was operated on. A thick indurated mass near the pylorus,lying along the lesser curvature,was seen. The ulcer was not excised,but posterior gastroenterostomy was done.

Case IX. Annie Jeffs,aged 39,a domestic servant, was admitted into the General Hospital,Birmingham, on Dec.2nd.,for severe pain and vomiting after food. Previous history was that she was under treatment for gastric ulcer two years before. There was no recurrence until six months before admission,when the pain and vomiting restarted,and they gradually got worse. An attack of haematemesis occurred six days before admission,and she then shewed signs of recent loss of blood. The abdomen was very tender in the epigastrium,with resistance of the upper parts of the recti muscles. Melaena was present.

Case X. Rose Yates, aged 31, domestic servant, was admitted into the General Hospital, Birmingham, on Oct. 16th., suffering from great prostration after an attack of haematemesis. Her previous history was that she had suffered from indigestion for five years, on and off. The pain was gnawing in character and would appear about 10-15 minutes after food, which would be vomited half-an-hour later, and this would cause relief. It was two days before admission that the attack of haematemesis occurred, which was repeated five times the day before admission. Her condition on admission was that of a pale, anaemic and semi-collapsed woman; pulse 86 per minute; temperature 98 F.. Shortly after admission she vomited blood again. The abdomen was sunken and the epigastrium tender, with resistance all over. In spite of medical treatment, pain and vomiting continued; so Mr. Heaton was asked to operate, which he did on Nov. 24th.. He found the stomach tied down posteriorly to the liver and pancreas, and there was a large irregular ulcer on the posterior wall of the stomach. Gastrojejunostomy was performed.

Case XI. John Yates, aged 26, occupation not stated, was admitted on June 27th., after severe and profuse haematemesis. The previous history was that for two years on and off he had had indigestion - that is, a lancinating pain immediately after food,

with vomiting,which relieved it. On admission, his face and body were blanched; his hands and feet were cold; his pulse was feeble - rate 88 per minute; his temperature was 98 F.. There was diffuse tenderness over the epigastrium and some resistance to the palpating hand. Melaena was present.

Case XII. Emma Fletcher,aged 35,was admitted into the General Hospital,Birmingham,on Sept.7th.,suffering from an-attack of haematemesis. Previous history was that for two years she had had dyspepsia _ that is,a severe gnawing pain in the epigastrium immediately after food,relieved by vomiting. Her condition on admission was that of a thin,pale and anaemic-looking woman with a pulse of 100 per minute and a temperature of 100.4 F.. Tenderness was marked over a small area in the epigastrium. There was some rigidity of the muscles on the left side. Dorsal tenderness was present also. Blood in the stools was obvious.

Case XIII. Annie Mason,aged 33,a barmaid,was admitted into the General Hospital,Birmingham,after severe haematemesis. The previous history was that two years before she used to suffer from severe gnawing pain immediately after food,with occasional vomiting. She had been quite free from gastric trouble until the attack of haematemesis a week before admission. The blood came after a continuous piercing pain for five hours. A second,

but less severe, attack of haematemesis occurred two days before admission. On examination, her tongue looked flabby and dirty, and her face was pale and "pinched." There was some tenderness on pressure in the epigastrium over a circumscribed area below the xiphoid process, and there was resistance of the upper belly of the left rectus. Melaena was present.

Case XIV. Annie Powell, aged 25, a brass-founder's assistant, was admitted into the General Hospital, Birmingham, on Feb. 14th., with a terrible pain in the stomach and back. The previous history was that for two years she had had indigestion, and she had had an attack of haematemesis. Periods of quiescence of gastric trouble had occurred. A month before admission a shooting pain started suddenly in the epigastrium after a meal, and it caused her to vomit five minutes after. The pain disappeared, and re-appeared with an attack of haematemesis two days before admission. The patient, on examination, looked pale and restless. There was some circum-scribed tenderness just below the xiphoid process. Muscular resistance was felt on the left side. Melaena was present.

Case XV. Samuel Hutson, aged 28, was admitted to the General Hospital, Birmingham, on Oct. 19th., after a severe haematemesis. Previous history shewed that pain and vomiting started six years

before,and would last 2-3 months at a time and then cease for two months or so. Three weeks before admission,he vomited blood three times; a week later the pain got considerably worse, almost "doubling him up." He could keep no food down. Eating a biscuit temporarily eased the pain. On admission,his tongue was dirty and furred. The abdomen was sunken,the costal arches thus being prominent. Tenderness in a circumscribed area, both in the epigastrium and dorsally,was present. Resistance of the left rectus in its upper part was felt. His pulse was 68 per minute - of good volume and regular; his temperature was 98 F.. Melaena was present.

Case XVI. Jane Garrington,aged 37,a domestic servant,was admitted into the General Hospital,Birmingham,on June 27th.,after an attack of haematemesis. Previous history shewed that she had suffered from indigestion for two years,on and off,but was free just before admission. On the day of admission, she was walking,when she felt faint and vomited a large quantity of blood,and so was taken to the hospital. Her stools had been black for a fortnight before she came to the hospital. Her condition on admission was that of a pallid and anaemic woman,with some tenderness over a small area in the epigastrium at the top of the xiphoid process. Tenderness was present also dorsally. There was no abdominal rigidity.

Case XVII. Horace Smith,aged 40,a tool maker,was admitted into the General Hospital,Birmingham,on July 15th.,after an attack of haematemesis. Previous history shewed that sixteen years before he had had severe pain in the stomach,lasting half-an-hour. Then it had come after every meal,food increasing it in severity. He had had to give up work on account of it. The pain was described as lancinating and sharp,and always in the epigastrium. The pain would return and then disappear. Vomiting commenced ten years before admission,occurred as often as six times a month,would last three months,and would then gradually disappear. It recommenced one month before admission,occurring three times a week, usually after food,but sometimes in the early morning. An hour before admission,whilst working,he vomited a quart of blood,and then fainted,and so was brought to hospital in a collapsed state. On examination, he was collapsed and pale. His pulse was 84 per minute and feeble,his temperature 96.8 F.. He complained of pain and diffuse tenderness under the left costal arch,as well as at the back. The stomach was not dilated. Melaena was present.

Case XVIII. John Thomas Speak,aged 51,was admitted into the General Hospital,Birmingham,on Feb.5th., from the out-patient department. The pre vious history was that eight years before he had had a "nervous breakdown,"associated with pain in the

stomach after food,lasting about six months. The pain returned three months before admission and appeared two hours after food. Vomiting of food commenced five weeks before admission,and of blood fourteen days before admission. The pain and vomiting were not continuous,but came and went. His condition was that of a man with deficient musculature and poor nutrition. He was pale and feeble-looking. His tongue was "dirty" and furred. There was marked localised epigastric tenderness and pain, and there was some resistance to palpation on the left side of the abdomen. This patient had sugar in the urine and blood in the stools.

These eighteen cases present a typical previous history of suffering from indigestion of varying time and intensity. All the ulcers progressed and became chronic. The slowness in the process of ulceration was seen when the vomiting of blood occurred. In the majority of instances, it was an attack of haematemesis that brought the patients into hospital. A number of these patients gave records of more than one attack of haematemesis.

(d)Acute Perforating Ulcer. This form,similar to the acute haemorrhagic,runs an initial latent course. Unexpectedly perforation occurs,which rapidly leads to death, by setting up an extensive general peritonitis.

As an example of this type of ulcer,I quote the case of William Lewis,aged 32,who was admitted

into the General Hospital,Birmingham,on May 10th.,suffering from a perforated gastric ulcer. The previous history was that he had had slight attacks of indigestion,but they had not been bad enough for him to stop work. Suddenly,on the day of admission,he experienced a violent pain in the stomach,which prevented him from working. He did not vomit. On examination,the patient wore an anxious expression. His pulse was 140 per minute and his temperature 100 F.. The epigastrium was tender to the touch and rigid. The percussion note in the left epigastric region was impaired. Mr.Barnes operated,and as soon as the abdomen was opened,the stomach contents gushed out. After exploring,he found a large ulcer,one inch in diameter,on the lesser curvature,about 2½ inches from the pylorus. The edges were cleanly cut. Extending towards the pylorus,was a large irregular ulcerated surface. There was a little peritonitis in the neighbourhood of the ulcer,and well marked perihepatitis. There was a large quantity of fluid in both hypochondria. The ulcer was directly closed by three silk sutures,the stomach wall was stitched over,and the abdomen then closed. I had two other cases,but these will be discussed under subphrenic abscess.

(e) Cachectic Form. This type of ulcer is a very chronic one. Frequent emesis, recurrent attacks of pain,lack of nourishment, and increasing emaciation

are typical of this variety. The patient is pallid and sick-looking, with sunken cheeks and hollow eyes - a condition more suggestive of carcinoma. This condition is often associated with advanced gastrectasis and the late stages of chronic hypersecretion when associated with an ulcer.

(f) Gastralgo-dyspeptic Form. In this type, dyspeptic symptoms predominate, and suggest chronic gastric catarrh. The usual complaint is one of pain, occurring irregularly after food, and discomfort. The stomach region is sometimes sensitive to pressure. There may be occasional vomiting. In these cases there is hypochondria, associated with absence of mucus.

I had sixteen cases illustrative of this form, and shall give particulars of them.

Case I. Charles Hunt, aged 30, a driller, was admitted to the General Hospital, Birmingham, on Jan. 4th., suffering from pain in the left side. The previous history was that the symptoms had started six months before, and pain, the most marked one, had never disappeared. The pain occurred 10-15 minutes after food usually, and sometimes it was so severe that he had to give up work. He vomited food occasionally - never blood. On admission, he was pale and shewed signs of having lost weight, and nutrition was impaired. The abdomen

moved freely. There was some slight tenderness in the epigastrium, to the left of the middle line.

Case II. Maud Sheldon, aged 33, domestic servant, was admitted into the General Hospital, Birmingham, on June 7th., from the out-patient department, with indigestion. The previous history was that she had suffered from dyspepsia for four years. The pain occurred a few minutes after food, and sometimes at night. It was gnawing in character, and used to radiate from "the pit of the stomach" to between the shoulders. She used to make herself vomit, so as to relieve the pain; otherwise she did not vomit. On admission, she looked thin, and had evidently lost weight. The tongue was furred. Some tenderness to pressure in the epigastrium was present, but no rigidity.

Case III. Maud Webber, aged 23, an assistant confectioner, was admitted on Nov.3rd. to the General Hospital, Birmingham, suffering from indigestion. Previous history shewed that it had lasted nine months and shewed no likelihood of improvement. There was considerable pain immediately after food, which caused her to vomit about once a day. This lasted perhaps a week. Then a period of quiescence would come, after which the pain and vomiting would return, the pain each time getting worse and the vomiting increasing in frequency. On admission, the patient was pale and shewed loss of fat. Nutrition was poor. The tongue shewed the indentation of the

teeth,and was white. The abdomen was scaphoid and a little rigid. Some epigastric tenderness was present,but no pain.

Case IV. Charles Burke,aged 44,a hotel porter,was admitted on Aug.2nd.to the General Hospital,Birmingham,with symptoms of indigestion. The previous history was that for ten years he had had pain in the stomach. It commenced as a slight discomfort after food; then actual pain was felt,until at times it became almost unbearable. The pain was well marked at night. He occasionally vomited,and when he did so,it caused relief. He had been living on milk and soda for a month before admission. His condition was that of a fairly well developed and nourished man. The tongue was furred. The abdomen was tender on the left side,but not painful. No rigidity was present.

Case V. Florence Bullock,aged 23,a domestic servant was admitted to the General Hospital,Birmingham,on Aug.4th.,complaining of pain after food. The previous history was that the symptom of pain had commenced four months before, and had been getting worse every day. Vomiting had commenced a fortnight before admission, and it relieved the pain. The condition of the patient was that of a pale and pasty woman. The tongue was furred. There were pain - slight only - and tenderness in the epigastrium,but no rigidity.

Case VI. Lucy Gilbert, aged 25, a housemaid, was admitted into the General Hospital, Birmingham, with severe pain and vomiting. Previous history shewed that, two years before, she had begun to have pain after food - usually 5-10 minutes after - but no vomiting. A year before this second admission, she had been treated as an internal patient for the same symptoms, and had been discharged cured. The symptoms recurred two months before re-admission, after she had been free for ten months. The pain was then paroxysmal and cutting after each meal and during the night. She vomited occasionally. On admission she looked thin and pale. Her tongue was dirty. There was some abdominal tenderness at a point midway in a line joining the umbilicus to the anterior inferior iliac spine on the left side, but there was no rigidity or pain.

Case VII. Kate Karnam, aged 18, a housemaid, was admitted to the General Hospital, Birmingham, on April 6th., suffering from pain in the stomach, heartburn and vomiting. The previous history was that pain after food had suddenly started one-and-a-half years before, and had grown worse and become stabbing in character, so that she had to go to bed. The pain used to last one hour. At that time she used to vomit after most meals, which produced relief. She had had these attacks on and off up to the time of admission. On admission, her tongue was seen to to be furred, and her teeth were bad. There was

slight tenderness in the epigastrium. Otherwise there was no abdominal discomfort.

Case VIII. Lilian Babington,aged 26,a pen worker, was admitted to the General Hospital,Birmingham,on Sept.8th. for pain and vomiting after food. The previous history was that these symptoms had started suddenly six years before as an acute agonising pain after food. Vomiting had commenced one day later. Periods of freedom,followed by dyspeptic symptoms, ensued,until an exceptionally painful attack brought her into hospital. Her condition was that of a pale anaemic woman. Her tongue was furred. There was some localised epigastric tenderness present below and to the left of the xiphoid process. No rigidity or actual pain was discovered.

Case IX. Edward Tomlinson,aged 54,a blacksmith, was admitted to the General Hospital,Birmingham,on June 7th.,for pain and vomiting after food. Previous history shewed that his complaint had commenced 15 months before. Pain,dull and aching,was experienced within two or three minutes after taking any solid food and usually lasted one hour. Vomiting started six months after the pain,and occurred only after a big meal. It relieved the pain,however. On examination,it was found that he was developed,but there was evidence of wasting. Some circumscribed tenderness was perceived under the left costal arch; otherwise the abdominal signs were negative.

Case X. Kate Rathbone, aged 25, a housemaid, was admitted on Nov.27th. to the General Hospital, Birmingham, for pain and vomiting immediately after food. Previous history shewed that, seven years before, she had been under treatment for gastric ulcer, which had been cured. Recurrence of the symptoms occurred nine months before admission. She then complained of a "heavy feeling" in the stomach, which developed into a gnawing pain before and after food. Vomiting afterwards followed, but did not produce entire relief. The pain was now present at night. On admission, the patient was anaemic and shewed signs of want of nutrition. There was a localised painful area on pressure under the xiphoid process, but no other abdominal sign.

Case XI. Lilian Spencer, aged 25, a pen worker, was admitted to the General Hospital, Birmingham, on April 12th., complaining of pain all over the abdomen and vomiting after each meal. Previous history was that three months before she had had indigestion very badly, which had become steadily worse, until now the pain occurred within five minutes of taking food. Vomiting had commenced about the same time as the pain, and took place half-an-hour after the pain had started. It sometimes relieved the pain. On admission, there was some tenderness under the left costal arch, but nothing more.

Case XII. Rose George, aged 35, a domestic servant, was admitted on Oct. 26th. to the General Hospital, Birmingham, complaining of attacks of pain, heartburn, giddiness, vomiting and headaches. Previous history showed that she had had symptoms of gastric ulcer 1½ years before, and had been cured. The complaint returned six weeks before admission, with pain and vomiting of food. There was flatulence continuously. On admission, the only abdominal sign was some localised epigastric tenderness just below and to the left of the xiphoid process.

Case XIII. Leonard Sigston, aged 36, was admitted to the General Hospital, Birmingham, on Dec. 19th., complaining of pain in the stomach, with vomiting. Previous history shewed that he had had three similar attacks, the first occurring about five years before, the next four years later, and the third three months later; and he was now admitted for recurrence of the symptoms. There was nothing in the physical signs of the abdomen that was outstanding. It was decided to operate, and Mr. Lucas performed a posterior gastroenterostomy, having found a large hard mass occupying the neighbourhood of the pylorus, and one inch from it.

Case XIV. Fanny Smith, aged 48, doing housework, was admitted on April 8th. to the General Hospital, Birmingham, complaining of pain and vomiting after food. The previous history was that six months before she had been under treatment for acute pain in the stomach after food, flatulence and occasional

vomiting. These symptoms had abated somewhat, but had returned with increased severity a week before admission, when her condition was that of a pale, anaemic-looking woman. Her tongue was flabby and coated. There was slight localised abdominal tenderness, on pressure only.

Case XV. Lizzie Green, aged 25, a domestic servant, was admitted on June 3rd., with the complaint of pain and vomiting after food. The previous history was that symptoms of gastric ulcer had become manifest three years before, but she had recovered and had remained well for two years, when the symptoms had returned and had been as bad as ever. Mr. Gamgee had been asked to operate, had done so, and had found a thickened mass on the lesser curvature, near the pylorus. She had then been discharged as cured. However, the pain returned three months before re-admission, commencing twenty minutes to half-an-hour after food. She vomited occasionally. As she did not improve, she was re-admitted. There was then diffuse tenderness and pain over the epigastrium. The pulse was good, regular, and at the rate of 84 per minute. The temperature was 99. The symptoms gradually subsided; so the patient improved.

Case XVI. James Sculthorpe, aged 34, a clerk, was admitted on April 16th. to the St. Giles and Bloomsbury Infirmary, London, complaining of pain and vomiting after food. Previous history shewed

that he had had an attack of haematemesis seven years before,from which he had made a satisfactory recovery. The recurrence of the old gastric symptoms of pain 1-2 hours after food and occasional vomiting occurred three months before admission. The vomit was large in quantity and contained mucus. On admission,it was seen that he was not much emaciated and his nutrition was fair. The tongue was dry,but not coated. There was marked pain on pressure over the appendix,and tenderness in the epigastrium. There were some shooting pains in both groins,radiating upwards to the umbilicus. There was some slight rigidity of the upper part of the abdomen. Percussion shewed that there was no enlargement of the stomach. Areas of urticaria-like weals(linear erythema) were visible round the umbilicus. He stated that the eruption used to appear over different parts of the body,and would last 2-3 days and then disappear. No cause could be assigned to the appearance of this erythema,occurring in localised patches. The forehead,extensor aspects of the forearms,hands,legs and ankles,as well as the neck,were the parts affected.

In this list it will be observed that there is a preponderance of female patients. All of these patients had some localising tenderness referable to the epigastrium. All shewed a progressive chronic course.

(g) Stenotic Form. This occurs in the process of healing, causing thereby constriction of the stomach cavity. The stenosis may occur at variable points, depending on the position of the ulcer. This ulcer is usually annular, or nearly so. This type produces dilatation of the stomach, as well as a condition termed "hour-glass stomach." In the latter condition, the ulcer, as it heals, contracts and divides the stomach into two compartments, which communicate through the stenosed ring.

I had seven cases of this type, all shewing some degree of dilatation but no "hour-glass" formation.

Case I. Caroline Ward, aged 47, doing housework, was admitted on March 20th. to the General Hospital, Birmingham, from the out-patient department. Previous history shewed that she had had indigestion for two years almost continuously. The symptoms became more aggravated six months before admission. The pain was shooting and severe and appeared after most meals, frequently lasting throughout the day. Vomiting was occasional, but the material was large in quantity. Flatulence worried her a good deal. On admission, it was seen that the upper portion of the abdomen was a little fuller than usual. Percussion was marked. The lower border of the stomach reached out a little below the umbilicus, its left border the anterior axillary line. Its fundus was as high as the fifth interspace. Palpation elicited some

splashing.

Case II. Beatrice Dodd, aged 20, a cycle lamp worker, was admitted into the General Hospital, Birmingham, on Dec. 11th., for pain in the stomach and between the shoulders. The previous history was that she had had pain and vomiting lasting two years and up to the time of admission, the pain occurring half-an-hour after food and remaining the whole day. When she vomited, which she did occasionally about one hour after food, the material was large in quantity and very acid. Flatulence was frequent. On admission, there was some tenderness over stomach area. Percussion brought out the lower border of the stomach to the level of the umbilicus, the outer border to the anterior axillary line, and the fundus in the fifth interspace. Splashing was obtained easily.

Case III. John Hughes, aged 27, a carman, was admitted to the General Hospital, Birmingham, on Dec. 14th., with return of old symptoms of gastric ulcer. Previous history shewed that two years before he had been affected with pain and vomiting after food, for which he had been treated as an in-patient. He was discharged cured, but returned to hospital three months later with the same symptoms as before, which made him seek re-admission. He said the pain disappeared on his lying down, and that vomiting was occasional, but large in quantity. On admission, some fulness was detected above the umbilicus. The abdomen was flaccid. The percussion note was a little tympanitic

and defines the lower border of the stomach to be at the umbilicus: some splashing is elicited. This patient was operated on by Mr. Heaton who found the stomach dilated and containing an indurated ulcer on the posterios surface near the pylorus. Posterior gastroenterotomy was done.

Case IV. Alfred Kershaw, aged 29, a mechanic, was admitted on March 24th to the General Hospital, Birmingham, complaining of pain and vomiting after food. Previous history was that for ten years he had suffered from indigestion with periods free from gastric trouble. For the two years previous to admission the pain had been almost continuous, commencing after breakfast and persisting throughout the day. Food always made it worse. He vomited occasionally and in large quantities. On admission the abdomen was somewhat fuller in its upper part: the percussion note was tympanitic and defined the lower border as lying two inches below the umbilicus, the left border as searching the middle axiliary line, and the fundus as lying in the fifth space. Palpation elicited loud splashing.

Case V. William Peters, aged 39, beerhouse keeper, was admitted on August 11th complaining of pains in the stomach and vomiting. Previous history showed that he had complained first of gastric symptoms eight years ago, when pain, followed by vomiting, was present after every meal. He was treated on and off

for two years,but each time there was a relapse. He then remained well for three years, when the old symptoms recurred. At that time his stomach was washed out, and after this he remained well for 18 months, then thepain and vomiting returned. Six months previous to admission the pain recommenced with increasing severity: the vomiting had stopped, but he used to make himself vomit to relieve the pain. On admission it was seen that the abdomen was fuller than it ought to be and gave a tympanitic note. The lower border was found by percussion to be at a point half way between the umbilicus and the pubis; the left border reacted almost to the posterior axiliary line, the fundus remained at the level of the 5th rib. On inflation the stomach stood out with prominence and it was possible to see fine peristalsis from left to right. Splashing was loud. He complained of pain just to the left of and on a level with the umbilicus. It was decided to operate on him on August 25th and Mr. Heaton found adhesions to the omentum only: the stomach side of the pyloric orifice was stenosed and hardened with a double ring of contraction, the seat of chronic ulceration. The stomach was enormously dilated and thin walled. Posterior gastroenterostomy was performed.

Case VI. Annie Freeman, aged 29, cook, was admitted to the General Hospital, on November 4th, complaining of pain and vomiting. Previous history pointed out that she had had an attack of haematemesis five years before from which she made a good recovery.

The symptoms however, had returned one year before but she was never quite free from pain or vomiting. Lying in bed with medical attention gave but temporary improvement. On admission there were signs of loss of weight and nutrition: the percussion note of the upper abdomen was unusually tympanitic: the lower border of the stomach reached to the umbilicus, the left border of the mid-axillary line, and the fundus as high as the 4th interspace. Splashing was elicited. There was no epigastric tenderness.

Case VII. Gertrude Milnes, aged 27, a housemaid, was admitted on July 25th to the General Hospital, complaining of pain and vomiting. For two years on and off, she had been attending the out-patient department for sudden pain and vomiting after food. The knife-like pain started in the pit of the stomach and radiated to the back between the shoulders, and always increased by food but relieved by vomiting. Flatulence was invariably present. On admission the abdomen appeared quite healthy: percussion note somewhat tympanitic: percussion defined the lower border just below the umbilicus, the left border reached the mid-axillary line and the fundus was as high as the 4th space. She experienced some tenderness, 1¾ inches below the xiphoid process, as well as dorsally over the 10th dorsal spine.

These 7 cases go to show that ulcers situated in the region of the pylorus, after healing have contracted

and narrowed the outlet to the stomach; the food, in consequence, remains longer in that organ; and its weight and the obstruction to its evacuation into the duodenum have produced dilatation.

Analysis of Stomach Contents - Removed through the Stomach-Tube.

It has been stated that there is no reason why the stomach-tube should not be passed after a recent gastric haemorrhage. Those in favour of this procedure argue that the passage of the soft tube is less harmful than the accompanying vomiting. Of course if the diagnosis of "ulcer" is positively established there is no need to pass the tube. The tube may produce a haematemesis or a perforation. Where the analysis of the stomach-contents has been performed, it reveals high percentages of hydrochloric acid, as much as 0.3% to 0.35% at the height of digestion. In about half the cases, the quantity of hydrochloric acid is normal, in others it is diminished. It has been shewn that in some cases during fasting the stomach contains hydrochloric acid — the so-called hypersecretion of Reichmann.

The secretion of pepsin does not undergo much alteration; but is diminished in chronic ulcers.

The motor functions of the stomach are not interfered with unless there be dilatation. When the stomach is dilated, there is bacterial decomposition with the formation of gases such as nitrogen, oxygen, carbonic acid, marsh gas, hydrogen, and occasionally sulphuretted hydrogen.

The methods of analysing the stomach contents, enumerated below, were those used at the General Hospital, Birmingham, by me and the results obtained were by those means.

The usual procedure adopted for the calculation of the stomach contents is to give a test meal. This meal is given on an empty stomach in the morning (say 8.0 a.m.) and consists of ½oz. of toast and a cup of weak tea (10 fluid oz.). It is then siphoned off an hour later by means of a soft stomach-tube. This test breakfast is recommended by Ewald. There are several other kinds of "meals", bearing the inventors' names, but the one used at the General Hospital, Birmingham, was Ewald's. After the withdrawal of this meal, another meal may be given consisting of 6 oz. chopped meat, 4 oz. bread, a glassful of water and some currants. This meal is siphoned off five hours later and gives us the information respecting the digestive and motor powers of the stomach. Any impairment is thus discovered by the presence of partly digested meat or currants. The healthy stomach is empty at the end of 5 hours.

The technique used in the introduction of the stomach-tube is simple. Certain points must be attended to before the passing of the tube, such as removing any false teeth and excluding the possibility of the existence of an aneurysm. The manipulation should not be carried out after a recent haematemesis.

Choose a long, smooth, red rubber stomach tube - at least a yard long - previously sterilised. Make a mark on it 18 inches from the "eye", so as to serve as a guide when the "eye" is in the stomach cavity. Fit the other end with a glass funnel. Insert in the middle of the tube a short piece of glass

tubing so as to act as an index to the progress of the withdrawal of the contents. The patient sits down with his head bent forward, facing the physician. He opens his mouth but does not protrude the tongue. The tube, previously moistened with warm water, is passed in the middle line along the dorsum of the tongue until it reaches its root, when, if the patient is now instructed to swallow, the tube will glide into the upper end of the aesophagus which grasps it. He proceeds to "swallow" the tube until it reaches the stomach cavity. There may be a few spasms of the œsophagus which prevent the passage of the tube, but these pass off after a time. Sometimes diverticula are met with into which the tube passes. The danger of passing the tube into the larynx is averted if the manipulation is done correctly. So long as patients breathe deeply and at the same time are able to talk, you know the tube is in the œsophagus and not in the trachea.

When the tube reaches the stomach, the funnel is depressed below the level of the stomach. After a moment or two the stomach contents are evacuated and collected in a clean receptacle. Sometimes the "eye" gets choked up with large morsels of food, in which case no contents can escape. This necessitates the withdrawal of the tube and its reinsertion. Pressure in the epigastrium usually suffices to start the evacuation. If this be not successful, pour down the funnel some clean warm water, having previously pinched the tube lower down, so as not to include air: now relax it; this allows the imprisoned air to bubble up through the water. The water now passes down the tube but does

not enter the stomach, as the funnel is now depressed again. The water runs out and as it does so sucks up the contents, which are collected in a separate receptacle.

We are now in a position to analyse the stomach contents which have thus been siphoned off.

The macroscopic appearance of the gastric contents of a test breakfast, removed at the end of one hour, shows that it is of a watery and sometimes semi-fluid or pasty consistency, with a quantity of amylaceous residue. It smells strongly acid. There may be blood in the contents, in which case they are tinged red. Usually the colour is yellowish or straw-colour.

The fluid is next tested with blue litmus, which is turned red: this shows that it is acid. The acid present may be due to free or combined Hydrochloric acids or even organic acids, chiefly lactic, acetic and butyric. To see if any free hydrochloric acid exists, dip into the filtered gastric contents Congo red paper. If there is free hydrochloric acid present, then the paper is turned blue. This is a sure test. Another positive test is with G. Rümburg's reagent. This reagent consists of dissolving 2 grm. of phloroglucin and 1 grm. of vanillin in 30 c.c. of absolute alcohol. The method is to take 10 drops of the gastric contents and place in a porcelain capsule, and with it mix an equal quantity of the reagent. Heat gently over a flame, keeping the dish moderately cool by blowing on the surface. As soon as the fluid evaporates, a red zone appears at the periphery if any free hydrochloric acid is present. The combined acid and organic acids do not yield the red colour.

There is therefore free hydrochloric acid present in the contents.

The next step is to estimate the total acidity. The total acidity is caused by the following factors:- (1) Free hydrochloric acid: (2) Hydrochloric acid in combination: (3) Acid salts: (4) Organic acids.

The quantitative determination of free hydrochloric acid and total acidity can be made from the same specimen of stomach contents.

The presence of free hydrochloric acid by quantitative tests is first determined, then 10 c.c. of the filtrate are used to estimate the total acidity. In the burette place 1/10 normal sodium hydroxide solution, and allow to drop into a beaker containing 10 c.c. of filtered gastric contents. From time to time remove a drop of the mixture with a platinum loop to Congo red paper, which is stained a bluish violet at first. As soon as the change of colour in the Congo red paper becomes indistinct, read off the number on the burette. The number of cubic centimetres of decinormal sodium hydroxide solution used indicates the amount of free hydrochloric acid present. A normal amount of free hydrochloric acid present is 0.1 to 0.2%: if more than this, hyperacidity exists.

At this juncture phenolphthalein solution is added to the gastric contents and we continue to titrate as before until the colour of the solution is red. The total quantity of decinormal sodium hydroxide solution used from the beginning of the titration indicates the total acidity. The normal total acidity varies from 40 to 60. Riegel found that the maximum of free hydrochloric acid in gastric ulcer was 6.9%, when the total acidity

was 130. Free hydrochloric acid attains its maximum in from 180 - 200 minutes and total acidity in from 180 - 210 minutes. The presence of organic acids is next determined. These are not present in the usual case of gastric ulcer, unless there is concomitant dilatation of the stomach with fermentation.

Pepsin is usually secreted and not interfered with. Its presence can be proved by adding stomach contents to a test tube containing pieces of hard-boiled egg and kept at body temperature. Digestion of the egg occurs if pepsin is present.

The presence of rennin is revealed by adding a small quantity of gastric contents (previously neutralised) to some raw milk, keeping the mixture at body temperature for about a quarter of an hour. The milk solidifies if rennin is present.

In the cases in which gastric analysis was done, I found that free hydrochloric acid was present in all, but in no case was it excessive, nor was the total acidity outside the usual limits. I quote four cases as having a bearing on this point.

Case I. Maud Sheldon was a case of the gastralgo-dyspeptic type. An Ewald test breakfast was given at 8.0 a.m. and drawn off at 9.0 a.m. Free hydrochloric acid was demonstrated by Günzburg's method. The estimation of the hydrochloric acid contents was 0·2% and the total acidity was 45: no sarcinae or bacilli were found microscopically. A Riegel test dinner was given and the stomach contents drawn off 6 hours later. There was little or no residue: meat fibres and currants were not seen.

Case II. Caroline Ward was a case of the stenotic form. In her case there was slight dilatation. An Ewald test breakfast

was given and the contents drawn off an hour later and examined. Free hydrochloric acid was present and amounted to 0·2% and the total acidity was 75: the microscope revealed the presence of yeast cells, starch granules and a few long bacilli. A Riegel test dinner was also given, the contents being drawn off 6 hours later: the deposit contained undigested currants and measured 2 inches. It was acid from the presence of lactic acid. Under the microscope partially digested meat fibres, starch granules and fat were seen. There was no free hydrochloric acid.

Case III. Alfred Kershaw, aged 29, a case also belonging to the stenotic form of ulcer. An Ewald test breakfast was given him and drawn off in an hour's time. Free hydrochloric acid was present and amounted to 0·18% and the total acidity was 60. Starch granules and a few long bacilli were seen under the microscope. Later a Riegel dinner was given and drawn off 6 hours after. There were 2 inches of residue which contained currants and other solid material. On analysis it was found that it was slightly acid from the presence of lactic acid, but no free hydrochloric acid was present. Many long bacilli, sarcinae and yeast cells were seen under the microscope.

Case IV. John Hughes, aged 27, another case of the stenotic variety of ulcer, was given an Ewald test meal. The stomach contents were drawn off and examined an hour later. Free hydrochloric acid was present and amounted to 0·25% and the total acidity was 65. He was given a Riegel test dinner which was drawn off 6 hours later and 3 inches of acid deposit were collected: currants, starch granules were found. The acid was lactic acid, but there was no free hydrochloric acid.

Course of the Disease.

(a) The course is usually chronic and progressive, though many cases end in recovery (according to Cruveilhier, about 80%). There may be little or no after effects. Lebert states that the average duration of an ulcer is from 3 to 5 years, some even as long as 20 to 30 years. Other cases run a rapid course, depending on how soon the ulcer is rationally treated. Others again do not heal because patients continue to live on an inappropriate diet. So long as pain persists it must be assumed that the ulcer is not healed.

(b) Other cases recover and in the healing process cicatrisation occurs. As a result of this, dilatation of the stomach occurs or an hour-glass stomach is produced.

(c) Other cases, again, do not heal, but show a progressive ulceration. This type of patient considers himself cured when all symptoms have disappeared under a strict diet and rational treatment: but as soon as he changes his mode of living, new disturbances appear analogous to the ones before, even after the expiration of many weeks or months. The only symptom that seems to persist after the ulcer has healed is hyperacidity. Patients should be advised, in view of recurrence, to continue to live a rational life and not to think themselves cured until the lapse of a considerable time from the disappearance of all symptoms.

(d) Lastly there are cases in which malignancy is implanted in the ulcer. The frequency with which this occurs is stated by Lebert to be 9 of every 100 cases. It was first suggested by Cruveilhier in 1835 and later by Rokitanski in 1839. Since those times the idea has been gaining ground, until it is now stated by Graham that ulcer predisposes to cancer in 61%.

The malignant tumour arises from the edge of the ulcer where cicatrisation has occurred. The glandular loops thus enclosed proliferate with simultaneous alteration of the epithelial lining. The carcinomatous ulcer has a smooth, hard base, its edges are thick and round, the neighbouring tissue is friable, and has a tendency to form adhesions to surrounding organs notably the pancreas.

The symptoms produced by the grafting of cancer on to the ulcer are similar to those of the primary ulcer. The pain becomes more constant, though slightly less intense and assumes a dull aching quality. There is more nausea and vomiting. Tenderness has become less limited and more diffuse. Blood now appears more frequently. The appetite is lessened and the patient becomes languid, anaemic and nervous. Palpation may now elicit a gastric tumour which increases in size. The examination of the stomach contents is of little use for the distinction. As has been stated previously, the hyperacidity continues for some considerable time after the healing of an ulcer. Sticker (23) states that "it seems to be the rule that in those cases in which free hydrochloric acid is found in carcinoma of the stomach, the carcinoma will have developed from a round ulcer". The production of a gastrectasis

and the above symptom complex renders the diagnosis easier, especially if a long time elapses between the cessation of symptoms ascribed to the ulcer and the developement of the cancer.

The course of gastric ulcer as illustrated from my cases is best tabulated as follows:-

(See next page for table)

Course of 50 cases of Gastric Ulcer.

Number of cases			Duration of Symptoms
	Cured	28	6 to 3 months: 4 to 6 months: 4 to 1 year 6 to 2 years: 6 to 3 to 5 years: 2 to 6 or 7 years.
"	Relieved	10	5 to 5 years: 4 to 10 years; 2 to 16 year
"	Unrelieved	3	3 to 2 years.
"	With dilatation as a result.	7	4 to 2 years; 1 to 5 years: 1 to 8 years 1 to 10 years.
"	With malignancy implanted on to ulcer.	None	None
"	With complications recent haematemesis	7	1 to 10 days; 1 to 14 days; 1 to 1 month 1 to 6 weeks: 2 to 2 months; 1 to 10 weeks
"	With perforation & subphrenic abscess	2	1 to 3 years: 1 to 5 years:
"	Subjected to operation	11	6 to 3 years: 2 to 5 years: 2 to 6 years: 1 to 8 years.
"	That recurred after operation	3	3 recurred 2 years after operation.
"	That died:	5	1 to 10 days: 1 to 6 weeks: 1 to 2 months 1 to 2 years: 1 to 5 years.

N.B.

In the above figures 6 to 3 months means six cases with symptoms persisting for 3 months.

From above analysis it will be gathered that 56% were cured, which is a small percentage compared with Cruveilhuer's figure of 80%. But Bulstrode found that 82% had been cured, and 40% of these had relapsed. Schultze states that 53% are cured.

The figures that represent the duration of Symptoms are included under more than one heading.

For instance a case might be included amongst the "unrelieved ones", as well as under those with dilatation, so the figures are relative only.

It is intended to point out by the above table the duration of symptoms before, say, a subphrenic abseess occurs.

Complications.

Ulcer may lead to many complications, notably haemorrhage, perforation, adhesions to other organs, cicatricial stenosis and hour-glass stomach.

(a) Haemorrhage. This has been alluded to under symptomatology as haematemesis. But bleeding not infrequently occurs in the course of an ulcer, though there may be no symptoms of ulceration present. The cause of it is due to erosion of one of the large vessels constituting the gastric epiploic plexus, in the course of the spreading ulceration. The bleeding may be fatal, the patient dying before help can be summoned. This condition is comparatively rare. It is more usual for a violent haemorrhage, as much as 1 to 2 pints, to occur with symptoms of a grave secondary anaemia. These patients are very pale, extremities cold, pulse rapid and easily compressed, and are very nervous, almost afraid to breathe. Bleeding is apt to recur in a few days, but is not so severe. I have already given details of haemorrhage occurring suddenly, under the heading of "acute haemorrhagic ulcer", but to make the subject complete I shall mention one case only.

The case was that of Arthur Horton, a market porter, who was admitted suffering from profuse haematemesis on July 22nd. He had abdominal pain for 10 weeks prior to admission, but on July 22nd he suddenly felt faint and immediately vomited about a pint of blood. He was very pallid, the tongue coated, the

abdomen was sunken but moved with respiration: some epigastric tenderness was felt in gentle palpation: the pulse was feeble and counted 84 per minute: 97·5°F. was his temperature. After treatment with salines and morphia he improved, but a repetition of the haematemesis occurred on the third day, and again twice on the fourth day: his condition was extremely grave, marked signs of anaemia and exhaustion were present, and despite energetic treatment he died. This case points out how fatal a haematemesis may be, and the likelihood of recurrences.

(b) Perforation. This is one of the most serious of the complications of ulcer of the stomach. It has been stated by many authors that perforation occurs in about 6·5% of all cases of gastric ulcer. Lebert's statistics show 3·5%: Fenwick's 5·5%.

As I have already stated, perforation is more liable to occur when the ulcer is situated on the anterior wall of the stomach, because that portion of the stomach is very mobile and furthermore there is less tendency to the formation of adhesions. The occurrence is common in young females, though relatively more common in males, since there are fewer cases of gastric ulcer in the male.

According to Brinton, 70% of all perforations are on the anterior wall. Again it is usually acute ulcers, situated on the anterior wall, that perforate, whereas chronic ones, situated on the posterior wall, tend to form adhesions. Perforation may be multiple and occurs in about 20%.

Perforation may occur without any previous symptoms though it is more usual to find that local pain and vomiting or even

haematemesis have been present. If the ulcer is situated on the anterior wall and perforation occurs, the stomach contents pass into the general peritoneal cavity, producing diffuse general peritonitis. If the ulcer is situated on the posterior wall and perforation occurs, the stomach contents pass into the smaller sac of the peritoneum. If the ulcer is situated close to the duodenum, the extravesation of stomach contents passes into the cellular tissue posteriorly, or burrows along the ascending colon or even about the kidney.

Perforation may occur without any particular cause, though it usually follows one of the following :- violent exertion, ingestion of a large meal, or a blow on the stomach.

Moynihan divides perforations into acute, subacute, and chronic. In the acute cases, which are very severe, the symptoms are sudden and the leak is opened to the peritoneum and the signs are general from the beginning. In these cases the ulcer is situated on the anterior wall.

The subacute cases are those in which the stomach is empty and the perforation small. In these cases there are slight adhesions, accompanied by severe tenderness and pain increased on movement, and occurring a few days before the ulcer perforates.

The chronis cases are those in which adhesions have ~~been~~ formed and in which perforation may occur. Partial or general peritonitis may ensue, and adhesions form between the stomach and adjacent organs: or a subphrenic abscess may result.

The course of a perforated ulcer is variable. In the acute perforative ulcer general peritonitis that supervenes may heal spontaneously by the formation of fibrin, closing the perforation.

It is more usual however for recovery to take place in the subacute or chronic varieties. Allowing for these rare cases where spontaneous healing does take place, we see a very high mortality -rate from perusal of statistics. These show that 95% of the cases, untreated surgically, die: that 25% of the cases operated on within 12 hours of rupture die: that 63% of cases operated on within 12 to 24 hours after rupture die. It is essential, therefore, to treat the case immediately after the diagnosis is established.

If the patient does not die, extension with the formation of adhesions to adjacent organs results. The perforation into the liver and pancreas may occur with abscess formation. Subphrenic abscess may occur. Catarrhal cholangitis from obstructive jaundice by pressure on the ducts is sometimes seen. Perisplenitis, pylephlebitis or involvement of the gall bladder are other sequelae. Invasion of the pleura through the diaphragm with resulting pneumothorax has occurred. Tillman collected 12 such cases which were all fatal. A similar invasion may take place into the pericardium and even into the left ventricle of the heart. Pick reports 10 such invasions of the pericardium. In the more chronic cases the pus may "point" some distance from the seat of the ulcer; such a one is reported by Bertholdstein who found the colon and pelvis of kidney contain gastric contents and pus.

The <u>symptoms and signs of a perforation</u> are characteristic. The onset is acute: the patient complains of a sudden violent pain in the abdomen, referred to the epigastrium at first, then diffused.

The pain may be continuous or suggestive of colic. Along with the pain are the symptoms of collapse: the face is anxious and pinched: and there is great restlessness in some cases: sometimes the signs of collapse clear up only to return more grave. The pulse is quickened, the temperature falls immediately after perforation, but rises later when peritonitis supervenes: the tongue is dry and the patient complains of thirst: vomiting is an inconstant symptom, though retching is more usual: urine is diminished in quantity, and micturition later becomes painful when peritonitis is well advanced. The abdomen may be flat and tense, sometimes is distended: in a few cases it is retracted: the muscles are rigid especially the upper bellies of both recti: there is diminished mobility: these last two signs are of the greatest significance. There is tenderness chiefly in the upper abdominal zone (epigastrium and hypochondrium), though not infrequently it is situated in the right iliac fossa. Percussion note is tympanitic, with diminution in liver dulness and possibly splenic from the accumulation of gas between liver and diaphragm. (This does not occur if adhesions exist between stomach and liver). Auscultation over the diaphragm may elicit a friction rub, and sometimes gurgling can be heard over the perforation if it is located.

If the patient has not died from collapse, which occurs in about 4% of cases, symptoms of peritonitis set in. The temperature rises to 102° or 103°: the pulse becomes harder and "wiry": breathing

more shallow: retching and hiccup follow, finally death from exhaustion or collapse.

I have already reported the case of William Lewis (under the heading of "acute perforating ulcer") and it is needless to report it again. The signs and symptoms of my patient are those discussed here. I have two other cases belonging to the chronic ulcer that perforated with the production of a subphrenic abscess which will be given in detail under "subphrenic abscess".

A Subphrenic Abscess may be produced as a result of perforation of ulcer of stomach, leading to the formation of an encapsulated abscess. It is usually situated on the left side. The abscess as a rule contains air as well as fluid. It is important to demonstrate the presence of an exudate in the lower portion of the thorax, when all pulmonary signs are absent. To determine this, it is necessary to make out the existence of vesicular breathing, and that the boundary of the exudate extends downwards on deep inspiration. At the same time the thorax on the afflicted side is not very ectatic, and the interspaces are scarcely obliterated. The lower portion of the thorax usually bulges. As a rule the heart is pushed upward slightly, whereas the liver is pushed down further into the abdomen, occasionally as far as the umbilicus. Percussion usually reveals, on the affected side, normal lung resonance; below it is a tympanitic note corresponding to the escaped air, and lower still dulness from the escape of pus. Some subphrenic abscesses do not contain air: these make diagnosis more difficult. Frequently inflammation is set up in the pleura lying on the diaphragm, giving rise to a pleuritic rub and later

an exudate. The differential points to be made out between an exudate in the thorax, e.g. an empyema, and a subphrenic abscess on the left side are these: (1) The patient lies on his back in subphrenic abscess, whereas in empyema he will lie on the affected side (left). (2) Violent pain, especially in the epigastrium and hypochondrium (left). (3) Pain and stiffness of the back when the patient attempts to sit up. (4) Pain on belching. (5) Oedema of lower lateral and posterior portions of thoracic wall extending to lumbar region.

A subphrenic abscess may terminate:-

(1) by perforating through the diaphragm into the lung with expectoration of the pus;

(2) by perforating through the skin with the formation of a gastro-cutaneous fistula;

(3) by perforating transverse colon with production of diarrhoea and pus in the stools;

(4) by perforating into the pleura with the production of a pyothorax;

(5) by perforating into the abdominal cavity, causing general peritonitis with subsequent death.

I had in my series two cases of subphrenic abscess which I detail below.

Case I. Bridget Holand, aged 32, doing house work, was admitted to the General Hospital, Birmingham, on Jan. 24th, 1911, complaining of a very severe sharp pain in the abdomen. Previous history showed that she had pain and vomiting directly after food for 5 years on and off. A month before admission she was seized

with a sharp pain in the stomach which passed up to the ensiform cartilage and then travelled back to the shoulder blades. It had disappeared for 2 weeks, only to reappear on the day of admission. She never vomited blood. On admission, the abdomen moved a little restrainedly with respiration; there was epigastric and dorsal tenderness to pressure; & some rigidity of the upper parts of the recti muscles. A blood count showed that there were 15,000 white cells per c.mm.: the liver dulness was not obliterated, but reached the costal arch: splenic dulness unaltered. The pulse was 92 per minute, strong and regular: temperature 98°F. The patient seemed well for two days, but on the third day the pulse increased to 108 per minute, and the temperature to 101°F: there was severe epigastric and dorsal pain: she rejected 5 ounces of milk: liver dulness unaltered. After three days the temperature, pulse and severe abdominal symptoms subsided. Five days later, however, there was a small area of marked tenderness under the left 6th costal cartilage; the abdomen was resistant to pressure, though moved easily with respiration. She complained of no actual pain, and there was no vomiting. She felt well for the next 5 weeks, always however complaining of some epigastric tenderness. No discomfort was felt after solids: vomiting never occurred. During this time she gained 2½ lbs. On March 5th she was discharged relieved after 6 weeks' residence. She was readmitted on April 28th (7 weeks later) with recurrence of the same severe abdominal pain. The left half of the abdomen was rigid, taut and tender: the pulse was 86, strong and full: temperature 95°F.: a leucocytosis was taken and enumerated 16,600

per c.mm. On the day following Mr. Heaton operated and found a thickened area in the lesser curvature of the stomach with fresh adhesions to the liver: no gastro-enterostomy was done. She recovered from the operation and improved well and was able to take solids. Then the ulcer showed signs of leakage, when the abdomen was again opened in the right hypochondrium, as the pain was chiefly over the gall bladder, and from the exploration nothing definite was found. Two days later, June 5th, she died.

The post-mortem revealed the fact that there was a median healed operation scar and an open one in the right hypochondrium: the lower lobe of the right lung was completely collapsed owing to the pressure upwards of the liver. There was an abscess in its lower portion. The pericardium contained turbid fluid and lymph. The portal vein at its entry into the right lobe of the liver contained a small attached clot and beyond this it was lost in an abscess cavity. On the posterior surface of the stomach near the pylorus was a scar of a healed ulcer attached externally to the anterior surface of the pancreas. Anteriorly a partially healed ulcer led by a small probe pointed orifice to a small abscess cavity lying between the stomach and the liver: the stomach was adherent to the liver all around. The liver was enlarged and its right lobe was riddled with abscess cavities. The operation wound in the right hypochondrium opened one of the abscesses, but not large enough to drain: the gall bladder contained two small calculi. This then was a case of Portal pyaemia with a subphrenic abscess originating from a leaking chronic gastric ulcer.

Case II. Robert Poynter, aged 38, general labourer, admitted to the St. Giles and Bloomsbury Infirmary on June 16th, 1913, complaining of pain in "the pit of the stomach" 20 minutes after food. Previous history was that three years before he was an inpatient of the same institution, suffering from attacks of haematemesis. He then had abdominal tenderness and some fulness of the upper part of the abdomen. Melaena was present for some time. He was discharged 3 months later as cured. However the symptoms of pain recurred 2 months before admission. There had been occasional vomiting which relieved the pain. His tongue was furred: tenderness on pressure was elicited over a large area with the xiphoid as its centre: some resistance was present in this same area: occasional splashing: no enlargement of the liver: stomach outline vague. The temperature was remittent and the pulse was usually between 80 and 90 per minute. A week after admission he experienced a very sudden short sharp pain in the stomach, but no physical signs of urgency were observed. Two days later he complained of a throbbing pain in an area extending from the tip of the xiphoid to the umbilicus: it was not increased on taking milk: he felt thirsty: there was occasional flatulence but no vomiting. Tongue was dry and furred. I noticed the abdomen in its upper portion suddenly swelling, which did not cause any distress or dyspnoea. The thoracic veins were distended: left and right hypochrondria, epigastrium and umbilical areas were full and tympanitic. The apex beat was not displaced. Percussion of this large tympanitic area revealed its boundaries as follows: inferiorly a finger's

breadth below the umbilicus, mid-axillary line to the left, the superior border in 5th interspace: to the right it reached the right nipple line prolonged downwards. Palpation showed considerable resistance over this tympanitic area. Auscultation revealed some friction over the left lower ribs, especially the 9th. Lower liver border was replaced by tympanitic note which was continuous with the tympanitic area just described. A stomach tube was cautiously passed and I drew off a large quantity of foul smelling material. The pulse was 88 per minute and the temperature 99°F. The diagnosis of a slowly leaking gastric ulcer was made. The day after the tympanitic area was less prominent: the liver dulness was further obliterated: some dulness was elicited in the right hypochondrium: pleuritic rub was increased and louder than before on the left side. Pulse 104 per minute: the temperature 99°F. It was now decided to operate, and Mr. Clayton Greene opened the abdomen and found the stomach, omentum and transverse colon matted together: a small perforation was seen on the anterior wall of the stomach, which indicated the size of the ulcer: the abscess was drained, and nothing further attempted, as his condition was bad. He made a good recovery from this primary operation, but declined any further operative measures. So he was discharged a month later quite free from pain or vomiting. These two cases indicate the necessity of being watchful for the appearance of leakage from the ulcer. In Poynter's case the acute sudden pain he experienced a week after admission was the sign of rupture.

(c) Adhesions to other organs: Perigastric adhesions are common in the chronic gastric ulcer, which produce symptoms that give a clue to the diagnosis of the complication. The sites of adhesions are about the lesser curvature in front and behind in the middle of the organ: at cardiac and pyloric orifices: the adhesions are dense and firm fibrous bands extending to the pancreas, liver, spleen, transverse colon and mesentery: the commonest being pancreas alone. liver alone and both pancreas and liver.

The symptoms vary and depend on the extent, degree and location of adhesions, as well as on the neighbouring viscera implicated. The commonest symptoms are pain after food, especially of a dragging kind, chiefly about the pyloric orifice: always worse after a big meal: increased during exercise and by the erect posture. If dilatation of the organ has been produced, vomiting large quantities of matter takes place which frequently contains an excess of hydrochloric acid. There may be some loss of flesh. Tenderness on pressure is elicited over the epigastrium. Sometimes a definite mass is felt, which corresponds to the matting together of the viscera: or the mass may be situated under the liver and fixed.

Briefly the differential points are: a healed ulcer or it may not be healed and of long standing: some gastrectasis: localised tenderness with pain shooting to the shoulder or breast: the influence of posture and sudden exertion:

periodical febrile attacks of short duration: occasional jaundice without hepatic disease: gastric analysis gives no distinctive results: the palpation of a definite mass in connection with the stomach. I have already mentioned cases in which adhesions were found at the operation or autopsy. I had fourteen cases with this complication and to avoid repetition I shall instance one case only.

Rose Yates had complained of dragging pains in the stomach after food for five years associated with vomiting. She was admitted prostrated from an attack of haemetemesis from which she recovered but the pain and vomiting persisted. To alleviate this condition it was decided to operate. At the operation it was found the stomach was tied down posteriorly to the liver and pancreas and that the ulcer was situated in the posterior wall. Her symptoms disappeared and she was discharged cured.

(d) Cicatricial Stenosis: After the formation of scar on a chronic ulcer, the scar tissue begins to contract and narrow the lumen of the organ. Wherever the ulcer is, especially if extensive, there is some contraction and resulting distortion. The commonest sites of stenosis are at either orifice - pyloric and cardiac - and any point in the body of the stomach usually nearer the pyloric side than the cardiac. As ulceration near the cardiac orifice is seldom met with, stenosis at that point is, in consequence, rare. It is more usual to

find pyloric stenosis which causes dilatation of the stomach. With dilatation motor insufficiency occurs and symptoms relative to those conditions arise. When stenosis occurs in the body the result is the production of hour glass contraction of the stomach. I had seven cases with cicatricial stenosis: which were described under the heading of " Stenotic type of ulcer" and need no repetition.

(e) Hour-glass Contraction of the Stomach. This consists of a contraction anywhere between the cardiac and pyloric orifices dividing the stomach into two compartments with an inter-communicating aperture. Robson and Moynihan reported forty eight of which forty one were due to ulcer. As a rule the pyloric half is smaller than the cardiac. The symptoms of this condition are sometimes definite, other times indefinite. There is usually some complaint of pain after meals, vomiting, emaciation and the presence of a tumour due to scarring of the old ulcer. It has been associated with symptoms many years. The diagnosis is usually made from adopting the following procedure:

(i) In lavage of the stomach, all the fluid fails to return.

(ii) After lavage has apparently thoroughly cleansed the stomach, a quantity of very foul smelling and dirty fluid returns.

(iii) After thoroughly emptying the stomach succussion note (splashing) is obtainable.

(iv) Distension of the stomach with air or gas demonstrates

the existence of two pouches separated by a narrow furrow. The stethoscope can elicit the passage of food through the constriction by the gurgling and sizzling noises produced.

(v) A bismuth meal is administered after a certain period X - Rays are passed showing on the fluorescent screen the constriction of the stomach.

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Diagnosis.

From the foregoing statements it can be seen how difficult the diagnosis of gastric ulcer may be. There may be no symptoms at all: one may get the symptom complex: one get a variety of symptoms, possibly only one of which is absolutely distinctive of ulceration.

There are three cardinal symptoms, which, if all are present, make the diagnosis positive, but absence of any one makes the diagnosis doubtful. Firstly: Pain in the region of the stomach occurring in paroxysms after food, and felt in a circumscribed area by pressure, in addition to hyperaesthesia. Secondly: Vomiting of blood or food. Thirdly: Increased values of hydrochloric acid.

Other symptoms such as waterbrash, heartburn, and occasional vomiting are less important.

Not infrequently pain is the only symptom though the diagnosis can be made if the pain recurs regularly at the height of digestion (half to one hour after food), if confined to the gastric region and if there are circumscribed painful pressure points. The pain is burning or stinging, frequently radiating to the heart or lung region or even into the arms. The pain or tenderness may be so severe as to prevent female patients wearing corsets. The most frequent direction in which the pain shoots is towards the vertibral column usually from the seventh to twelfth vertebrae.

The pain in the majority of cases is closely connected with

the ingestion of food, both as to its quality and quantity. Still there are patients who complain of pain at night time or even on an empty stomach. The time of onset of pain after food is ½ hour to ¾ hour, yet it may occur an hour or more afterwards, and this usually points to the ulcer being situated at the pylonis An important point in this connection is the regularity with which the pain comes on. The patient will state that after every meal he has an attack ½ or ¾ hour afterwards. If the pain commences whilst the patient is eating or immediately after a meal, the condition indicates an ulcer at the cardia.

The attacks of pain usually continue for some time, an hour or more and cease, possibly with vomiting or after the emptying of the stomach. Lying on the left side increases the painfulness while absolute rest and abstinence from food decreases it.

The appetite may be normal or increased at one time, absent at another.

The vomiting occurs at the acme of an attack and relieves the pain. It is produced as the result of spasms. The vomitus is usually well digested and mixed with mucus and characterised by a strongly acid both in odour and in reaction.

Haematemesis frequently occurs in a condition of apparent health. It may appear during the night, sleeping or walking. The vomit may be bright red or the colour of coffee grounds. The blood is partially coagulated when it is passed, and soon coagulates in the receptacle afterwards. This is a point of dis-

tinction with haemoptysis in which the blood is frothy and bright red and remains fluid for some time.

Melaena will occur if there has been haemorrhage from the ulcer. The stools are tar coloured and somewhat constipated. The evidence of blood in the stools is shewn by the tests already described as it is well known that iron, bismuth, coffee and chocolate render the stools dark brown or black.

Tenderness or Painful Pressure Points, when situated in the epigastric and dorsal areas, are of some clinical value by themselves, more so if associated with pain and vomiting. These points are circumscribed as I have indicated before. The dorsal point, when present, is of the greatest diagnostic value even though there is no epigastric painful point. The epigastric point is characterised by its intensity which serves to differentiate it from other gastric disorders.

Hyperacidity is, of itself, scarcely sufficient to warrant a case being labelled "ulcer" though, when present, especially combined with the other symptoms serves as an important agent for differential diagnosis.

Diagnosis of the Typical and Atypical Ulcer. The diagnosis therefore of a typical ulcer is easy when there are the above mentioned symptoms and signs. All cases however do not present such classical information. It is safe to pronounce the existence of ulcer if the characteristic ulcer pain and haematemesis are present. But it is difficult to diagnose the Atypical Form. Here the symptoms are varied and are not characteristic

or there may be ~~more~~ none as I have emphasised previously. Boas puts great stress on the value of the seat and the manner of occurence of pain as an aid to diagnosis. There may be uncertain dyspeptic symptoms prevalent which confuse rather than help in the diagnosis. The absence of haematemesis or melaena does not render the diagnosis any easier, in fact makes it almost impossible. There are usually associated with gastric ulcer, symptoms of nervous dyspepsia which act as a source of mistaken diagnosis. Physical signs of the abdomen have been enumerated but are not very definite. Levenet[38] determines the outline of the stomach, which is often very difficult, by giving the patient an effervescing mixture and examining him in the erect posture. Direct auscultation i.e. placing the ear directly to the stomach area, is then done. A sizzling noise is heard over the stomach area. I have not tried this method.

Another method is the radioscopic one of Cerné and Delaforge[24] who state it is particularly of value in those cases not presenting the usual symptom complex: These observers do not fill the stomach with the necessary bismuth test meal but watch the stomach in process of filling. For this they use bismuth suspended in cream. They believe this method provokes a better physiological excitation of the stomach than the usual mucilaginous suspension of bismuth. By a series of diagrams indicating the radioscopic shadow the authors are able to show the value of this method, not only in simple ulcer without complications, but

also in the latent ulcer, and in the various forms of gastric stenosis, and in gastric ulcer with adhesions in which the stomach's position is often greatly altered.

Radiological Diagnosis of Penetrating Gastric Ulcer.

Handek (25) has observed that such an ulcer gives rise to a special appearance on the X-ray screen, taking the form of a patch or streak of bismuth, isolated from the rest of the meal, or branching out from it, and usually to be found at the lesser curvature and middle part of the stomach. At the summit of this patch thus isolated, there is a bubble of gas. Further indications are the retention of bismuth for a considerable period in this region and the immobility of the bismuth patch, uninfluenced even by palpation or pressure. This observer further states that this method can be used as a differential diagnosis to cancer of the stomach. In cancer there is a defect in the bismuth shadow, due to the projection of the tumour into the lumen of the stomach. Furthermore he has never seen the bubble of gas which is usually imprisoned in the diverticulum of a penetrating ulcer.

The Diagnosis of Acute and Chronic Ulcers.

It is very probable that chronic gastric ulcers arise from the acute variety, because transitional conditions between the two may be found. Acute ulcers may be considerably delayed in its time of healing, and may also gradually extend. The tendency for the acute variety is to heal rapidly as well as to recur. An acute ulcer, as I have previously pointed out, is commonly latent and announces its presence by a sudden perforation, a profuse haematemesis or by pain and vomiting, with or without haemorrhage. I have mentioned instances occuring in my records of these. The relapses that occur in acute ulcers are frequently diagnosed as relapses in chronic cases. Points of difference do occur and are as follows: The pain, in acute ulcers, is not so severe as in chronic ones, and not so commonly paroxysmal and independant of food, neither is it so liable to be continuous. Vomiting is commoner in the chronic forms. In the acute variety the attacks of haematemesis are more liable to be profuse, commence and to cease suddenly. Hyperacidity occurs frequently in chronic ulcer: in acute ulcer where the few estimations have been made there is normal acidity or subacidity. Wasting is seen only in chronic cases. Long intervals between attacks are in favour of acute ulcer: on the contrary diminishing lengths of intervals and a condition of more or less constant pain and vomiting are characteristic of chronic ulcers. Acute ulcer is commoner in the young, especially women.

The diagnosis of my series of fifty cases was based on the following points which are tabulated below.

	Typical Pain.	Atypical Pain.	Vomiting	No Vomiting	Haematemesis recent	Tenderness Epigastric & Dorsal	Epigastric only
No. of cases	46	4	24	6	7	12	36

	Melaene	A history of Haematemesis	Hyperaesthesia
No. of cases	25	13	28

The table points out how few cases exhibit signs or symptoms which are not peculiar to those of gastric ulcer. There was little doubt in the majority of my cases where the diagnosis could not be made absolute.

DIFFERENTIAL DIAGNOSIS.

Many diseases have to be considered in connection with the differential diagnosis, the most important being chronic gastritis, gastralgia, simple hyperchlorhydria, gastric crisis in tabes, malignant disease, gall stones, pancreatic disease, gastrostaxis,(?) anaemic dyspepsia in girls, and vicarious haemorrhage, duodenal ulcer, embolism of superior mesenteric artery. Haematemesis, the one symptom on which a diagnosis is generally formed, is apt to be misleading, as it may occur after the swallowing of blood coming from the gums, nasopharynx or tonsils: bleeding from the mucous membrane of the stomach may occur from conditions other than gastric ulcer, such as in haemophilia, splenic anaemia, scurvy and purpura: it has been noted after gall stones and after abdominal operations: (Purves)(26): it has occurred in acute and chronic gastritis and in dilatation of the stomach: rupture of dilated and varicose vessels at the oesophageal orifice from cirrhosis of the liver and chronic heart disease produces haematemesis. Hence haematemesis can be misleading if its significance is not duly considered.

(a) Hyperaemia may cause haematemesis. Watson reported a case of this character. The patient, a female, suffered from gastric haemorrhage since her

fourteenth year, the bleeding occurring at the menstrual period. After she was married, the attacks of bleeding stopped during pregnancy and lactation but appeared at all other times.

(b) Anaemic Dyspepsia in chlorotic girls and young women produces symptoms which are sometimes difficult to distinguish from those of gastric ulcer. The symptoms are pain in the epigastrium and vomiting. Almost everything placed in the stomach produces pain, which is relieved by vomiting. They are probably due to (1) hyperaesthesia of the gastric mucous membrane, which in turn is due to (2) dilatation of the heart. This process is frequently associated with severe pain and tenderness referred to two parts of the body, (1) the skin and muscles under the left breast and (2) gastric mucous membrane. The mucous membrane is so sensitive, though quite healthy, that it causes the stomach to empty itself. Any exercise likely to stretch the cardiac muscle will in turn cause pain and vomiting. For instance, running upstairs will bring on the pain and vomiting. A.P.Beddard (27) emphasises these facts in the PRACTITIONER. The pain in these cases is not related to food at all, but is brought on by sudden exertion or after being tired out. Furthermore, tenderness affects the whole area over which the stomach is represented, and is excessive.

In spite of this tenderness, rigidity is frequently absent. Again, percussion of the heart demonstrates the left border to be some distance outside the nipple line. Especially is this the case when skin tenderness is present.

(c) Haemorrhagic Erosions or Gastrostaxis. Erosions may arise from a small capillary haemorrhage into the mucosa or from acute inflammation of the lymphoid follicles. Nothing can be found anatomically, however. There are no means of differentiating such a condition from gastric ulcer, as the following symptoms show: burning pain occurring a quarter to three-quarters of an hour after food, of variable intensity and diffused over the stomach, without hyperaesthesia or dorsal tenderness: occasional vomiting of material which may contain altered blood: normal or subnormal acidity of the gastric contents and the presence of blood-stained fragments of mucosa in the water of washings from the empty stomach. These oozings, according to Hale White,(28) are neither vicarious nor hysterical, nor the early erosions of gastric ulcer. The cause is obscure. I shall give notes of one case that was admitted to the General Hospital, Birmingham. The patient, Alice Dowell, aged 42, was admitted on Dec.3rd, with haematemesis. Previous history was that 15 years before she had had a slight haematemesis, and 14 months before

admission she passed blood per rectum, but there was no haematemesis. Four days before admission she complained of pain between the shoulders and round the left side, and the following day there was some haematemesis and again the following day. Condition on admission: she was pale and thin, pulse 108 per minute, small, rapid and low tension; temperature 98° F. Abdomen was slightly tender in the left epigastrium but no actual pain was experienced. Soon after admission she vomited 6 ounces of blood, and the result of enema disclosed the presence of blood in the stools. She remained 5½ weeks in hospital and was discharged cured. There was no pain or vomiting during her stay. Treatment adopted was the application of an ice bag to epigastrium, ice and iced water by the mouth, nutrient enemata, and after two days diluted milk for 7 days, then bread and milk, and finally chicken after 20 days. Iron and magnesium sulphate were given to counteract the anaemia and constipation.

(d) Embolism or thrombosis of the superior mesenteric artery is another condition, though rare, that simulates perforation of the stomach. It is accompanied by very severe epigastric pain and tenderness with vomiting and collapse. Haemorrhage is common. This condition is best illustrated by a case, of which I had one. Paul Swift, aged 39, was admitted with collapse

from the casualty department of the General Hospital on April 6th, complaining of acute pain in the abdomen, and also of having vomited a large quantity of blood. Previous history was, he had suffered from pain after food in the stomach for three weeks. Three days before admission he vomited all food for the first time, whilst the following day he vomited blood, about three pints. On the day of admission he was very collapsed, as he had just got over another attack of haematemesis: he was almost pulseless: the abdomen was distended and extremely tender all over. The surgeons refused to operate, as he was too bad. The following day he died. The autopsy revealed that the portal vein and its branches with the liver were distended with a blood clot: similarly the tributaries of the mesenteric veins were distended with blood clot, in each case formed before death. The stomach and duodenum were filled with blood: mucous coat was healthy with no sign of ulceration: veins of jejunum and ileum were distended and contained blood clot: blood was in the lumen of the jejunum and ileum: no gangrene present. Large colon contained blood: mesenteric arteries healthy: peritoneum contained blood and free fluid.

(e) Rupture of Oesophageal Varix. The differentiation of gastric and oesophageal haemorrhages is often quite difficult. The causes likely to lead to oesophageal bleeding are cancer, ulcer, varicosities and passive congestion. Blood from the oesophagus is frequently not

mixed with food and is dark red in colour. Age and sex may be of help, as varices are more frequently found in men above 40. Repeated and usually copious gastric haemorrhages take place from the varicose vessels. I had one case that occurred at the General Hospital, Birmingham, of which I shall give some details.

The patient, Mathew Hatton, aged 59, was admitted under Mr. Barling on Sept. 5th, with the complaint of vomiting blood. On admission the patient presented a thin and wasted appearance with signs of considerable loss of blood. The pulse was 72 per minute, regular but small. The temperature was 96.6 F.. He lay in bed in a listless, sleepy condition. At intervals he vomited blood about 1 to 2 pints. He did not complain of pain or tenderness, and rigidity of the abdomen was absent. He was treated with rectal salines every 4 hours, hypodermic injection of morphia ($\frac{1}{4}$ gr.), an ice bag to the epigastrium, ergotinine also given hypodermically. Nothing was given by the mouth. Later in the day he became clammy and unconscious, and died.

Postmortem notes. The aorta and hepatic artery showed wide spread atheroma: the portal vein was thickened. The oesophagus revealed many dilated, longitudinally-placed veins situated in the submucous tissue of lower $\frac{2}{3}$. One of them had ruptured, the opening being situated $\frac{3}{4}$ inch from the cardiac orifice of the stomach.

The mucous membrane of the stomach was chronically inflamed: a quantity of clotted and fluid blood was present in the organ. The liver was much enlarged and was a fine specimen of multilobular cirrhosis: the capsule was thickened.

(f) Vicarious Menstruation. Haemorrhage at the time of menstruation may occur from the stomach. It is rare and frequently associated with hysteria.. Only prolonged observation of such a patient will clear up the diagnosis. The vessel walls have usually undergone fatty degeneration.

(g) Chronic Gastritis. The symptoms of this disease are: the pain is not localised and not so severe as in ulcer: it is not increased by pressure and/is generally accompanied by flatulence, distension or discomfort for hours after food: vomiting occurs at irregular intervals, the vomit containing mucus organic acids sometimes and diminished quantity of hydrochloric acid: haematemesis is rare. The appetite is diminished. The tongue/is coated, flabby, and foul. The chief causes are oral sepsis, abuse of tobacco and alcohol, taking irritating food, chronic renal and heart disease, and cirrhosis of the liver. The appetite is capricious and pulsation in the epigastrium is common. I shall refer to three

cases of chronic gastritis.

Case I. Sarah Ann Elson, aged 39, was admitted to the General Hospital, Birmingham, with the complaint of pains in the stomach after meals. Previous history pointed out that this had been going on for 4½ years, though she had suffered from general debility and a feeling of being bloated and miserable for some time. Pain commenced 4½ years ago, starting in the lower part of the abdomen, in the chest and between the shoulders after every meal. The pain commenced almost as soon as she had taken food, and might last until the next meal, which did not relieve the pain. She frequently went to bed, which produced temporary relief. She vomited once in fourteen days, and this always occurred after she had taken food. She never vomited blood or even passed blood in her motions. Her bowels were very constipated. She had always had a good appetite but been afraid to eat. Her condition showed that she was fairly well nourished and apparently in good health. Her teeth were not very good. The abdomen was soft and no tenderness was elicited. The treatment adopted was milk and barley water, equal parts 3ß hourly and continued for two days; then bread and milk were substituted. Solids were given after 6 days.

Therapeutically a mixture of bismuth and soda was given three times daily. She left hospital very little improved.

Case II. Kate Murray, aged 50, was admitted to the General Hospital, Birmingham, with the complaint of pain in the abdomen and vomiting. These symptoms started 2 years before with pain a quarter of an hour after food, which persisted for a variable period. She latterly used to vomit, which relieved the pain. These symptoms used to disappear for a month, only to return intensified. Recently, however, she had been almost in constant pain so that she dared not eat any solid food. Vomiting was not frequent, but it relieved the pain. The vomit was always slimy but never contained blood. Bowels constipated. Her condition showed that she had lost weight and wasted; a thin, spare woman; no tenderness in the abdomen but a sense of discomfort.

The diet adopted was milk and barley water 3ß of each hourly, which was gradually increased every day by 1 oz. for 3 days. Then bread and milk for three days, when minced mutton was added. Solids were given after six days. A mixture of bismuth and soda was given before meals. She was discharged relieved.

Case III. Charles Burke, aged 46, was

admitted to the General Hospital, Birmingham, with the complaint of pain in the side of the abdomen. Previous history showed that these symptoms had commenced two years before with pain appearing one hour after taking food. He frequently had the pain at night. He never vomited but he made himself vomit so as to relieve the pain. The vomit was slimy and acid. He was an out-patient for about 11 years on and off, with chronic indigestion. His condition showed that he was well developed and fairly well nourished. Some slight tenderness elicited a deep palpation of the epigastrium; otherwise the abdomen was quite healthy. His diet was firstly of pure milk, 3 pints daily for 2 days, when bread and milk was added. Five days later solids were ordered. A mixture of bismuth and soda was given. He was discharged relieved.

(h) Gastralgia. In the differential diagnosis of this complaint, the following points are of value. (1) The pain is not dependent on ingestion of food and occurs periodically with great severity. (2) Algetic points are found over coeliac and solar plexuses with a tenderness which is smaller if compared with gastric ulcer. (3) The existence of hysteria, anaemia, uterine or ovarian trouble would help in confirming the diagnosis. (4) Haematemesis

is never seen. (5) The craving for certain types of food is frequent, and the appetite is capricious. I have two examples of gastralgia to which I shall refer.

Case I. Frederick Birch, aged 34, was admitted to the General Hospital, Birmingham, complaining of pain in the abdomen one to two hours after food. This pain commenced 11 months ago. He did not vomit. The appetite had been fairly good, but he had been forced to live on a light diet. He suffered from flatulency and constipation. Owing to the severity of the symptoms, Mr.Heaton was asked to operate on him. At the operation, stomach, duodenum, gall bladder and liver were found perfectly healthy, so the abdomen was closed. He was discharged eventually relieved.

Case II. Florence Kinsey, aged 18, was admitted to the General Hospital, Birmingham, with the complaint of pain in the abdomen and vomiting. These symptoms had lasted 7 weeks. Her illness commenced as a "bilious" attack, with pain and vomiting, coming on suddenly and as quickly disappearing, only to return a few days later, until now the pain was continuous. The vomiting was not so bad. There was no blood in the vomit. She was told to rest in bed, but the pain was still agonising. Her condition was that of a pale girl with blotchy face. The abdomen was not tender, but some distension of the upper half

was present. Under dietetic and therapeutic treatment she improved and was discharged relieved.

(k) Simple hyperchlorhydria. In this condition there is pain, waterbrash from one to two hours after food, with occasional vomiting, which affords relief from pain. The vomit shows an increase in free hydrochloric acid but no blood. The pain is greatest in the epigastrium though diffused over the whole stomach area, and there is rarely tenderness on pressure unless pyloric spasm exists; otherwise pressure relieves the pain. Dorsal point of tenderness and epigastric cutaneous hyperaesthesia are frequently absent. In hyperchlorhydria the pain reaches its maximum at the height of digestion, somewhat later than ulcer, and is relieved by food for the time being. There is great relief from the use of albuminous foods combined with alkaline treatment.

(l) Gastric Crises. Some cases of locomotor ataxia may be associated with severe attacks of abdominal pain and vomiting, which may simulate ulcer. These crises occur independently of food, and very rarely is haematemesis present. The pain is felt over a wider area, and is relieved by vomiting. The main feature of bad cases is the uncontrollable vomiting without febrile disturbance. The attacks may last from a few hours to several days. There is usually ample evidence of the presence of tabes dorsalis,

but occasionally the general symptoms are slight. The following case illustrates these points. The patient, Thomas Lewis, age 34, was admitted to the General Hospital, Birmingham, on July 12th, with the complaint of pain in the stomach. A luetic infection occurred 9 years ago and the pains in the stomach had lasted six years. He would wake in the morning feeling sick, and later would vomit a yellowish liquid. The vomit contained little partially digested food. He would vomit several times daily. Nothing relieved the pain or vomiting. He would feel quite well the next day. The next attack might occur 2 or 3 months later. Till 3 years ago the attacks became more frequent and lasted 2 or 3 days, even longer. Two years ago the attacks of pain and vomiting occurred every month and lasted from 3 to 8 days. One year ago the attacks appeared every 14 or 21 days. Just lately these attacks had come in 7 to 10 days, the vomiting lasting 2 days. The pain was always present in the upper part of the abdomen. Frequently pain was present only. Flatulence was frequently present a quarter of an hour after food. The vomit was translucent fluid with undigested particles. On examination of the abdomen, tenderness was marked out over an area extending from the right lumbar region, across the umbilicus to the left lumbar region. No tenderness

in the epigastrium. There was firmness and rigidity of the abdomen. Apart from this the ordinary signs of tabes dorsalis were present, namely, Argyll Robertson pupil, absent knee jerks, ataxic gait, and Rombergism pain was present in the chest and back as well. Analgesia was present in the anterior surface of the trunk, extending from third rib to the umbilicus posteriorly from the spines of the scapulae to the buttocks and thighs /both legs:/ he could feel pain in both feet just below the fibula. Touch was present throughout the body. Morphia (gr.$\frac{1}{4}$) and Chloretone (gr.V) were administered to control the symptoms of pain and vomiting. He was discharged relieved on August 9th.

Crises of Hypersecretion in Gastric Ulcer.

Mathieu[29] points out that these crises are found principally in ulcers of the pyloric region. The more pronounced symptom is pain, though they may occur in haematemesis or melaena. In these cases there is the presence of a hyperchlorhydric residual fluid in the stomach in the morning. The vomited matter may be almost black in colour or simply of a brownish tinge. The pain is coincident with spasm and at first is relieved by taking food and alkalies, but finally resists treatment. If the ulcer is situated on the lesser curvature, there is the absence of the morning fluid with clapotage. A diagnosis has sometimes to be made from tabetic crises. The chief symptoms are identical in the main, but there

is no progressive dyspepsia in tabes in which the attack is sudden, as is also its termination. Further, there is no clapotage and no pain on pressure. These crises may have to be distinguished from the painful crises of pyloric stenosis of malignant origin. The hypersecretion is due to stasis; and further, the vomited material is of a foul and disagreeable odour. There is also progressive feebleness and loss of appetite in malignancy. The slighter forms are treated by complete rest and atropine subcutaneously. Sometimes pylorectomy or gastroenterostomy is done.

(m) Malignant disease in the form of carcinoma (though sarcoma is met with) produces some difficulty in the early stages. The following points must be noted for the differentiation of the two diseases. With regard to course, it is seen that gastric ulcer may extend for years, with remissions: strength of the patient is usually maintained in ulcer but not in cancer. Ulcer is an affection of younger and middle age, while cancer appears later and its duration is seldom longer than two years. As to pain: this symptom is usually absent, though there may be a dull feeling of pressure in the epigastrium: in ulcer, pain is more common and occurs synchronously with ingestion of food. Vomiting in ulcer appears early, usually one to two hours after food, but in cancer it is irregular and occurs usually after a greatly

protracted retention of stomach contents. Stomach contents vary, but as a rule, in ulcer, there is rapid digestion of food and the vomit is in a fine pultaceous mass, small in quantity, in which coarse remnants of meat are only exceptionally found. There is present free hydrochloric acid. In cancer the vomit is large and contains many coarse and undigested meat fibres: the smell is acid: the vomit is usually intensely acid, though frequently hydrochloric acid is absent, the acidity being due to the presence of organic acids, chiefly lactic acid. The vomiting of blood is less decisive, and is usually less in quantity in carcinoma. The presence of a tumour is not diagnostic of carcinoma, as cases of supposed cancer have been operated on and the tumour found to be an enormously thickened pylorus or an encapsulated peritonitic exudate, or a cicatricised thickening at the base of an ulcer, or a tumour of a neighbouring organ, or a gastrolith.. The absence of a swelling is usually regarded as a negative sign of cancer. If the tumour grows and cachexia develops and increases, and an examination of the stomach contents reveals a progressive reduction in the secretion of hydrochloric acid and in the peptic power of the juice and at the same time the presence of lactic acid, the diagnosis of carcinoma is very probable. The presence of Oppler-Boas bacillus is detected in the stomach contents of a cancerous patient

but not in gastric ulcer.

I shall give details of the following case of cancer of the stomach. The patient, Annie Hall, aged 46, was admitted to the General Hospital on Nov. 9th, complaining of pain in the stomach and vomiting. Previous history showed that for two years she suffered from chronic indigestion. The pain was never very bad but vomiting increased in severity. She rapidly lost weight. Occasionally she vomited a small quantity of blood. Her condition on admission showed that she was considerably emaciated and anaemic. The examination of the abdomen revealed that the muscles were wasted, and a lump was felt in the epigastrium below the costal margins and to the left of the mesial line. It was hard, firm and fixed. Some dilatation of the organ was present. It is worthy of note here that her mother and one sister died of cancer of the stomach. She remained 25 days in hospital, then she died. During her stay, she gradually got worse, the vomiting being excessive, and the pain increased. The postmortem notes were as follows:- The pyloric half of the stomach was involved in a large ulcerated friable carcinona, extending over the whole of the mucous membrane, being about 4 inches in extent laterally and involving the whole circumference. The growth extended right up to the pylorus. On the anterior aspect about the

middle was a hole due to perforation, which would admit a finger with ease. A purulent/adhesive exudate covered the anterior aspect all round the perforation. The peritoneum showed a general peritonitis in the upper abdomen, being shut off below by adhesions between the transverse colon and the anterior abdominal wall.

A new method has been adopted by Fabian for the differential diagnosis between gastric ulcer and cancer. Fabian (30) has repeated the experiments of Grafe and Rohmer with the ethereal extracts of the gastric contents. Gastric contents after a trial breakfast contain a substance which acts haemolytically in cases of carcinoma. Out of 28 cases, 23 gave haemolysis in gastric cancer. In the other five cases, all of which were advanced cases of carcinoma, no haemolysis was obtained. Lympho-sarcoma yielded positive haemolysis. In 5 out of 7 cases of gastric ulcer no haemolysis was obtained, nor was it obtained in other benign affections.

(n) Cholelithiasis. The distinction between this disease and gastric ulcer is easy, but difficult when the characteristic haematemesis and tar-coloured stools are absent on the one hand and biliary grit or gall stones on the other.

The pain in cholelithiasis is not in

connection with ingestion of food, and occurs when the diet is bland in as great intensity as when the food is difficult to digest. There is tolerance to all kinds of food, alternating with paroxysms of pain. The pain is paroxysmal and the pressure point is in the region of the gall bladder. Icterus is absent sometimes. Frequently the liver, especially the right lobe, is painful and enlarged. Fever may be present as a complication. The pain moderates toward the right shoulder in gall-stone colic.

(p) Duodenal Ulcer offers no certain diagnosis, differentiation being almost impossible from a gastric ulcer at the pylorus, but the following points help one. In duodenal ulcer the pain is felt in the region of the right parasternal line, in gastric ulcer at the pylorus it is more toward the middle line. If haemorrhage occurs, the blood is found in the stools. The pain appears as a rule much later in duodenal ulcer: dorsal painful pressure-points are absent. Ulcer in the duodenum is more frequent in males than in females. Vomiting is generally absent in duodenal ulcer; when present there is no relief from pain. The absence of haematemesis is also another important point in favour of duodenal ulcer. I shall give details of three cases that occurred at the General Hospital, Birmingham.

Case I. Catherine Smith, aged 36, was

admitted on December 23rd, complaining of pain and vomiting after food. Previous history was that 12 months before she had a very severe attack of pain in the right part of the abdomen just under the ribs. The pain was felt in the front as well as in the back, though less severe. Vomiting usually occurred 2 to 3 hours after food. Except for short periods she had pain daily. She was never jaundiced and her motions were never black. The abdomen moved freely and was somewhat rigid in its upper part to the right of the middle line. Tenderness was elicited about one inch to the right of the middle line below the xiphoid process. On the day following, Mr. Barling operated and found an ulcer, the size of a shilling, situated on the anterior well of the duodenum, about one inch from the pylorus. Posterior gastro enterostomy was performed.

Case II. Edward Capsey, aged38, was admitted on Nov. 23rd, complaining of pain in the abdomen and vomiting. Previous history was that 6 years before he suffered from indigestion, which disappeared after more food. The pain occurred 2 to 3 hours after food and was situated in the right hypochondriac and epigastric regions. Vomiting occurred several times a week. No attack of haematemesis was recorded. Flatulence was constant.

He said he had lost weight. Latterly the pain had been made worse by food. On examination the patient looked well and healthy. He complained of abdominal pain, and tenderness on pressure was discovered in the right hypochondrium. There was slight rigidity over the same area. Two days after admission, Mr. Barling operated and found a firm indurated cicatricised ulcer in the duodenum close to the pylorus and involving it. The stomach was slightly dilated from this pyloric stenosis. Posterior gastroenterostomy was done. He was discharged cured.

Case III. William A. Liggins, aged 30, an artificial toothmaker, was admitted on July 19th, complaining of pain in the stomach. Previous history showed that for 18 months he had had pain in the epigastrium, scalding in character, occurring two hours after food, relieved at first by food. It was accompanied by a sensation of fulness in the stomach and flatulence and vomiting of food occasionally. There had been intervals of freedom of symptoms for as long as three months. The present attack had lasted a month. There was no history of accumulative vomiting, no history of haematemesis or melaena. The patient looked pale and thin: his tongue was coated: abdomen was flaccid on the whole but slightly rigid in right part of epigastrium. There was tenderness on

pressure, one inch to the right of the mesial plane and one inch below the subcostal plane: the area of tenderness was distinctly localised. The percussion note was hyperresonant in the epigastric region. The day after admission he was operated on by Mr.Haslam, who found that there was an annular irregular thickened constriction of the duodenum about one inch from the pylorus. A posterior gastroenterostomy was performed. He was discharged cured after one month's residence.

(r) Pancreatic Disease. Acute pancreatitis is rarely diagnosed, as it simulates other acute abdominal lesions. It is characterised by severe epigastric pain and tenderness sudden in onset, and is accompanied by vomiting and swelling of the abdomen: collapse and occasional rigors invariably follow.

This disease has points in common with a perforation of a gastric ulcer; so I mention the diagnostic points, which are:- the disease occurs twice as often in men as in women (Bosanquet)(31): the patients are usually stout or fat people with a history of alcoholism or gall stones: jaundice or hepatic symptoms are frequently precursors or accompaniments of the disease. Sometimes there are preceding symptoms of an impacted gall stone with paroxysmal rigors, pain,

pyrexia and jaundice. The pain is of a "sudden prostrating agony": extreme restlessness and the abdominal position assumed is not that of peritonitis. Delirium is frequently present. Whilst I was resident at the Stanley Hospital, Liverpool, I had three cases of acute pancreatitis, which were not diagnosed before operation. The first case was a dock labourer, aged 35, who had been drinking heavily for 10 days and vomiting incessantly for 16 hours. He complained of intense paroxysmal epigastric pain; the abdomen was retracted and fixed, especially in the upper part; he was delirious and had a rapid thready pulse with abnormal temperature. The abdomen was opened by Mr. A. Evans, who saw that the under surface of the omentum was studded with white areas: the pancreas was a mass of necrotic tissue. The patient died in 6 hours. At the postmortem no calculi were found.

The second case was a woman aged 51, fat and of alcoholic habits, with a history of bilious attacks. She had been vomiting for several days and had intense pain in the upper part of the abdomen. There was no jaundice, no abdominal distension, but fixation of the upper part of the recti. The abdomen was opened by Mr. A. Evans, and white glistening plaques of fat necrosis on the omentum

were seen. The gall bladder contained numerous calculi. This patient recovered.

The third case, a healthy man of 52, with no biliary history, was suddenly seized with severe epigastric pain and frequent vomiting. He had obstinate constipation, followed by diarrhoea with green offensive stools: there was sugar in the urine. A month later he had another similar attack. This time he was operated upon. The abdomen was opened by Mr. A. Evans and extensive adhesions were seen and separated. The peritoneum was red and showed no fat necrosis. An irregular mass was felt behind the stomach and it was drained. The gall bladder was healthy. He improved for 3 days after, the sugar disappeared, but on the tenth day the patient became delirious and died.

PROGNOSIS. It is stated that ulcer of the stomach is more often fatal in men than women. Rest, which is so important for the cure, is more difficult to obtain for a long period. Brinton collected 234 instances of death from perforation and found that 160 were females and 74 were males. It must be remembered that females are affected in the proportion of 2 women to 1 male. He further states that deaths from haemorrhage are four times as frequent in males as in females. Lebert's statistics of fatal cases are 57 females to 41 males: Habershon's are 35 females to 28 males. In my series the proportion is 3 females to 2 males.

Further the liability to recurrence is more frequent in the male than in the female. Habershon had 91 cases of relapse out of a total of 201 cases, and of these 42 were males and 52 females. In my own cases 21 recurred; 7 were males, 14 were females.

Again, complications and sequelae are more common in males. Habershon had 14 males showing dilatation of the stomach and only four females. I had the following complications amongst my cases, which are best tabulated:-

Number of Cases.	Haematemesis	Perforation	Cicatricial Stenosis and Gastrectasis	Adhesions
	16	2	7	14

The relative proportion of the sexes affected is shewn thus:

Haematemesis		Perforation		Gastrectasis		Adhesions.	
Male	Female	Male	Female	Male	Female	Male	Female
5	11	1	1	3	4	8	6

It may be taken for granted that the course of the disease is more grave in the male sex than in the female.

Prognosis as to age is an important factor. According to Brinton the average age at which death occurs in the female patient is 27, and the risk gradually diminishes. In my cases the ages were 32, 35 and 40 years, which are much later than the average age.

In the male the risk is distributed over the whole period of life, being slightly greater below the age of 50. The ages in my 2 fatal cases were 26 and 39 years - much earlier than the average age. The prognosis of the disease in persons past middle life is more unfavourable than in those who possess the vigour and vitality of youth. Very young subjects are unable to resist a serious attack of gastric haemorrhage and perforation.

If the disease can be recognised at a sufficiently early stage (before haemorrhage takes place) and the patient be put on a suitable regimen, much time will be saved. It is very necessary to maintain the strength of the patient during active ulceration, and this is done by rectal nutrient enemata and stimulants. In an old person, this cannot be persevered with indefinitely but recourse must be made to feeding by the mouth at a date much sooner than with a young person. During and after convalescence, it is necessary to impress on the patient the necessity of avoiding foods likely to irritate the stomach. The longer a patient can be induced to live on a strict diet, the safer will he be from a recurrence.

The gravity of the disease is increased in a youthful person, in a weakened constitution and in an old person: the risk of perforation is greatest in middle life, especially in young females. In the male, there is a greater tendency to relapse, to the production of dilatation of the stomach and to a fatal ending. A second attack is of graver significance than the first. The presence of coincident maladies and cachexia in an old case of ulcer renders the outlook more grave.

Is it possible to tell if the ulcer is healed?

In general, it can be said that if the patient be free from digestive complaints, if pain be absent, especially after solid food, if epigastric tenderness have disappeared, and if no occult blood be found after repeated tests in the faeces, the conclusion is that the ulcer is healed. This is strengthened if it be the fact that the patient has put on weight and strength.

Welch states that 85% of patients are cured and 15% die: the cause of death was perforative peritonitis 6.5%; haemorrhage 3.5%; other complications 4 to 5%.

Débove and Rémond's figures are these: cured, 50%: deaths 50%, made up thus: perforative peritonitis, 13%; haemorrhage 5%; inanition, 5%; tuberculosis, 20%.

Ewald's figures are: cured 75%. Russell of Birmingham states that 42.6% were cured and 44.7% still suffered.

From my cases I found that 28 cases or 56% were cured and 5 cases or 10% died. The causes of death in my five cases were perforative peritonitis, two cases: haemorrhage, 3 cases.

According to Leube, 75% of all cases will be cured by 4 or 5 weeks treatment: he further states that from his experience 21.9% improve under treatment, and 1.6% are not relieved. Lebert

states the mortality rate to be 10%, made up as follows: 6⅔% from perforation into the peritoneal cavity, 3% from haemorrhage, and the rest from exhaustion.

Bulstrode's statistics of 500 cases taken from the London Hospital show that 18% were fatal, of which 10% were due to peritonitis, 2.5% to haemorrhage, 5.5% to other causes. He further mentioned that 40% of the 500 cases had relapses.

The earlier the patient is treated, the more favourable the prognosis. Many cases recover without any special treatment, and others suffer from relapses. Some cases have abdominal complications at a remote date, others die from inanition or haemorrhage.

TREATMENT

(a) General. All treatment should be directed towards aiding the process of healing, and to effect this it is necessary for the patient to undergo a rest cure. A wound cannot heal if it is continually being irritated and stretched. Hence it is important for the patient to leave his occupation alone, and if he persists in working, the cure becomes uncertain. This rest cure is strongly emphasised by such men as Cruveilhier, Wilson Fox, Ziemssen, and Leube. The difficulty associated with the rest cure is that it

is almost impossible to keep the stomach at rest functionally for a prolonged period.

The method of carrying out this rest cure was started by Donkin, who treated his cases by rectal alimentation. Previous to this, food by the mouth was interdicted only for a short time after a haemorrhage. (39) Donkin extended his treatment to 23 days and observed favourable results, as did Mc Call Anderson, (40) and Boas, (41) who adopted the same procedure. Riegel and Leube continue the rest cure exclusively for several days, and do it not only in recent but also in chronic cases.

The method adopted by Riegel and Leube is as follows:- The patient receives from two to four nutritive enemata daily for 8 or more days. At the end of that time a little food is allowed by the mouth. The patient is in bed all the time, not even allowed to rise to micturate or defaecate. As thirst is so very common, it is advisable to allow the patient small pieces of ice to melt in the mouth. Frequent washing of the mouth is necessary. This principle allows for the maintenance of the body during the healing of the ulcer. The patient's health and nervous system are well maintained, and the stomach is kept at rest. The teeth should receive regular attention so as to prevent a nidus for the growth of bacteria. Some antiseptic tooth powder and mouth wash must be habitually used. Boas, Ratjen and Rost

state that the rest cure yields the best results. The nutrient enemata consist chiefly of artificially digested food such as peptonised milk-gruel, peptone suppositories or enemas of raw eggs and beef tea with a little brandy. The following substances are the most useful constituents of nutrient enemata: pure glucose, powdered casein (plasmon, sanatogen) or peptones, alcohol and salt. The bulk of the enema should not exceed 10 ounces. Each constituent should not exceed a concentration of 10%. Before giving a nutrient enema, the rectum is washed out with tepid water.

The "rest cure" with modifications was adopted at the General Hospital, Birmingham. The method was to order from the beginning, in a case exhibiting severe gastric ulcer symptoms, salines and nutrient enemas per rectum for 3 days. These rectal injections were every alternate six hours. Before the enema was given a rectal wash-out was done. Nothing by the mouth was given during this period. The mouth was frequently washed out with an antiseptic fluid, to which was added a little lime water or lemon juice to allay any thirst if present. This was carried out also after attacks of haematemesis. The salines consisted of normal sodium chloride solution and were given slowly through a rectal tube and funnel. The nutrients given the same way consisted of milk, 4 oz.,

one egg, common salt ℥i and liquid pancreaticus ℥i After the nutrient had been predigested with the pancreatic fluid, it was then injected slowly at body temperature. This was done every 6 hours for 3 days. The cases that were treated this way numbered nine They were as follows:-

Case I. Rose Yates was admitted after severe haematemesis in a collapsed state: the pulse 80 per minute, and the temperature 97° F.. She was placed in the recumbent position, warm bottles were put in the bed and morphia N.V. was injected subcutaneously. A rectal saline consisting of normal saline, one pint and glucose ℥i was given, and followed six hours later by a rectal nutrient. These were given for 3 days, alternating every six hours. Then milk was given orally. Ice was given to her to suck.

Case II. James Dowling was admitted with much tenderness and pain in the abdomen. He was put in bed and salines, with glucose, and nutrient anemas given for five days, when these were stopped and oral feeding commenced.

Case III. William Reynaert was admitted after a severe and profuse haematemesis. He was pale and showed signs of having lost a large quantity of blood. He was given salines and nutrient enemas for seven days when he was operated upon. He was given

ice orally and Liq. Morphia subcutaneously when it was required.

Case IV. John Yates, was admitted after a profuse haematemesis and in a blanched condition. He was given rectal salines every 4 hours, and nutrients every 6 hours: ice to melt in his mouth: liq. morphia m.V and adrenalin solution m.XXX orally. The salines and nutrients were continued for 4 days, when oral feeding started.

Case V. John Thomas Speak was admitted with severe pain in the stomach and vomiting. On admission he was given rectal salines alternated with nutrient enemas every 6 hours for 7 days. Predigested meat ℥iv was added to the nutrient and given in the 24 hours. Occasional sips of water were allowed.

Case VI. Fanny Smith was admitted after having vomited blood. Rectal salines and nutrient enemas were given for three days, when oral feeding was begun.

Case VII. Annie Mason was admitted after a severe haematemesis. After a simple enema of soap and water on admission, a rectal saline was given and followed six hours later by a nutrient enema. This was continued for 5 days, when she was put on oral feeds.

Case VIII. Arthur Horton was admitted after a severe haematemesis. He was semi-collapsed. Rectal

salines were commenced immediately and continued 4 hourly. Liq. Morphia m.V was injected. Adrenalin chloride solution (1 in 1000) M.XXX was given by the mouth. Nutrient enemas were given, 6 hourly, and continued to his death (4 days later). Subcutaneous salines were given on the 3rd and 4th days, and on the latter day horse serum was prescribed. Adrenalin chloride 3i was repeated and strychnine m.VIII given hypodermically.

Case IX. Horace Smith was admitted after an attack of haematemesis. Rectal salines, 4 hourly, then 6 hourly, to alternate with nutrient enemas, were given for 3 days, when oral feeds were started.

All these cases have been mentioned before; their previous history and condition on admission have been given in detail. They were all in-patients of the General Hospital, Birmingham.

(b) Dietetic. Following a preliminary period of exclusive rectal feeding, feeding by the mouth is gradually established and milk has been chosen for the purpose since the days of Cruveilhier. Milk is alkaline, it therefore lessens the acidity of the gastric juice and is non-irritant to the stomach mucous membrane. It does not remain long in the stomach and does not call forth vigorous peristalsis of the stomach. Reichmann discovered that boiled milk left the stomach more rapidly

than unboiled milk, and that the lumps of casein are smaller in the case of boiled milk. The nutritive value of milk can be increased by adding milk powder (100 gm. to 1 litre) or one or more teaspoonfuls of condensed milk or nutrose. Some patients cannot take milk but would probably take buttermilk, which however, is less nourishing. Sour milk is taken in large quantities in South Germany.

It is advisable in starting on a diet of milk that it should be diluted with an equal quantity of lime, soda or Vichy water or albumin water. The mixture is given warm, though it may be taken ice-cold. Not more than 4 to 6 ounces should be taken at once, and from 3 to 4 pints may be given in 24 hours. If the patient does not tolerate diluted milk, the milk may be peptonised. And if tolerance to peptonised milk is not possible, then albumin water alone may be substituted, or small feeds of meat juice (Brand's, Valentine's). Fleiner recommends meat jelly made by boiling a chicken and some beef with calves' feet. Strauss (42) advises the administration of sugar solution during the day, say 60 gm. of sugar in 20% solution. This diminishes the secretion of gastric juice. Rolleston advises milk diet for 10 days.

As treatment proceeds it is necessary to increase the nutritive value of milk by adding cream, simple

farinaceous foods, such as gruel and peptones. After this the pa tient may take bread and milk, arrowroot or tapioca, Benger's food, blanc-mange, and finely scraped raw beef.

If the pain do not recur and there is reason to believe that cicatrisation is established, then the patient may pass on to solids such as boiled chicken, pigeon, fish, and eggs. The last articles of diet a patient is allowed are mutton, roast beef, veal, venison, pike, soups, leguminous vegetables, potatoes, and trout. By this time the patient is out of bed and taking gentle exercise. Salts are given during the rest cure, especially Carlsbad salts. There is no explanation why the salts are given except an empirical one. They are said to diminish the hyperacidity of the gastric juice. Hence Carlsbad water, Hunyadi Janos, or Aesculap are used as the chief aperients.

To summarise briefly the rest cure: The patient is instructed to remain in bed for 2 weeks at least. Hot poultices to the epigastrium are required if the pain is bad. He defaecates and urinates in bed. Every day before breakfast he should take Carlsbad water or salts (one or two teaspoonfuls dissolved in ¼ of a litre of warm water). The diet is exclusively of milk with the addition of albumin water or Vichy water. When milk cannot be borne, then small quantities of meat solution or meat jelly are allowed. Rectal alimentation is administered

exclusively for 8 or more days, especially so after a haemorrhage. Then the gradual increase of the quantity and kind of food is made until solids are well tolerated.

(43)

Lenhartz in 1901 at the Congress for Internal Medicine suggested a concentrated egg-albumin diet as being more suitable to combat hyperchlorhydria. Lenhartz keeps the patient at complete rest in bed for four weeks. For the first 14 days an ice bag is applied continuously to the stomach to prevent gaseous distension to obviate haemorrhage and to relieve pain. On the first day even if a recent haemorrhage has occurred, 7 to 10 ounces of iced milk are given in spoonfuls and 2 to 4 eggs, beaten up with a little sugar, and cooled by placing the cup in ice. He allows a little wine to be added if necessary. The food is supposed to prevent hypersecretion, which causes the pain and vomiting. Bismuth in 30 grain doses is administered twice or thrice daily for the first 10 days. The food is increased daily by $3\frac{1}{2}$ ounces of milk and one egg, so that at the end of the first week the patient is getting $1\frac{1}{2}$ pints of milk and 6 to 8 eggs. This amount is maintained for a further week. About the 6th day the patient takes 35 grammes of raw chopped meat, alone or stirred up with the eggs. He is now able to take some rice or gruel, and in the third week a light mixed diet is given, the meat being broiled. The bowels are not moved for the first week: in the second week moved

by injections of warm water and glycerine. Blaud's pills are prescribed at the end of the first week. Lenhartz advocates this method of feeding for three reasons; (1) continuous administration of concentrated albuminous food which will use up the hydrochloric acid and prevent its acting on the healing ulcer; (2) rapid increase of nourishment to repair lost strength and thus aid in healing the ulcer; (3) absolute rest in bed for four weeks and prevention of distension.

He states that his method shortens the duration of treatment, that pain and vomiting are relieved, that recurrent haemorrhages are less (only 6.4% had recurrent haemorrhages), that the good effects are lasting.

Senator (44) adopts the middle course and recommends a diet consisting of gelatin, fat and sugar. Cases admitted to hospital with recent haematemesis are placed on a diet of white gelatin and sugar, administered every one or two hours. Fresh butter and cream are also given in small quantities, frequently repeated. If there be no recurrence of haemorrhage, milk, beaten eggs, and scraped raw meat, are added.

W. Weinstrand (45) has given his results after adopting Leube's treatment, which consists of (1) rest - at least 10 days, (2) Carlsbad waters, (3) diet of milk, soups, soaked rusks, (4) poultices. When the patient is free from pain, nausea and vomiting, one can add boiled rice or semolina, eggs, then minced mutton. After 12

days it is possible to add vegetables and stewed fruit, and then pass to a mixed diet. He had 134 cases under this treatment, viz. those of 108 women and 26 men: 89 had haematemesis. All these terminated in recovery, except that of one old lady. After a haematemesis he withholds oral feeds, whereas Lenhartz gives 2 eggs and 200 grammes of milk. He has found that intravenous injection of 10% sodium chloride stops the haemorrhage, the results of the injection being, in fact, extraordinary. (46)

Singer has for the last four years carried out Lenhartz's treatment. He found that immediate feeding after a haematemesis could not be borne by the patients, so he resorted to rectal feeding. Otherwise he had good results from it. The patients put on weight surprisingly. He found rest in bed an essential factor in the cure. For a haemorrhage, Singer advocates a 5% or 10% solution of gelatin alba and adrenalin. Hypersecretion and hyperacidity were combated with a 1% or 2% solution of anaesthesin in mistura amygdaline. He prefers to give haemoglobin instead of iron salts to counteract the anaemia.

The milk diet, used in the General Hospital, Birmingham, for cases of gastric ulcer, is graduated in the following manner. It is commenced from the day of admission or after a severe haematemesis.

On the first day, (either after admission or a haematemesis), equal parts of milk and water, thus:-

1st day Milk ℥ss and Water (Albumin, barley, soda or lime) ℥ss hourly.
2nd " Milk ℥i and Water ℥i 2 hourly.
4th " Milk ℥ii and Water ℥ii 2 hourly.
5th " Milk ℥iv and Water ~~℥iii~~ ℥ii 2 hourly.
6th " Milk ℥iv and Water ℥ii 2 hourly.
7th " Milk ℥v and Water ℥ii 2 hourly.
8th " "Low" diet (Milk pudding and bread and milk).
10th " "Middle," (Milk pudding, bread and butter and bread and milk).
14th (if no pain) "Middle" and minced mutton.
18th " " ") Fish.
21st " " ") Varied.

This diet was the usual one; in some cases it was modified. Patients were able to get on to solid food in 18 days after admission.

In several cases the milk could not be tolerated, so it was prepeptonised or citrated (gr. iii of sodium citrate to each ounce of milk).

The following patients out of my series were treated with that diet with the progress and results appended.

Name of Patient	Milk Diet	Length of time in Hospital	Day of solid food.	Result.
Horace Smith	Citrated Milk	23 days	14th	Cured.
Gertrude Griffen		31 days	14th	Cured.
Florence Billock		27 days	13th	Cured.
Maud Webber,		26 days	15th	Cured.
Eleanor Holt	Milk diluted with barley water.	20 days	14th	Cured.
Nora Scorfield	Citrated	28 days	16th	Cured.
Annie Powell		21 days	15th	Cured.
Jane Garrington	Citrated	40 days	21st	Unrelieved.
Kate Karnam		20 days	14th	Cured.
Charles Burke		22 days	15th	Cured.
Caroline Ward	Peptonised	39 days	19th	Cured.
Beatrice Dodd	Citrated	28 days	18th	Cured.
Annie Freeman	Citrated	29 days	16th	Relieved.
Gertrude Milnes		25 days	15th	Relieved.
Edward Tomlinson		34 days	14th	Cured.
Annia Mason		32 days	18th	Cured.
Kate Rathbone		29 days	16th	Cured.
Fanny Brown	Citrated	33 days	20th	Relieved.
Maud Sheldon	Milk diluted with albumin then barley water	43 days	22nd	Relieved.
Lilian Spencer		19 days	14th	Cured.
Annie Jeffs	Citrated	31 days	17th	Cured.
Norah Pearson	Citrated	41 days	22nd	Unrelieved.
Lucy Gilbert	Undiluted	36 days	16th	Unrelieved.

Name of Patient	Milk Diet	Length of time in Hospital	Day of solid food.	Result.
Beatrice Figgures		22 days	13th	Cured
Alfred Kershaw		20 days	14th	Relieved.
Robert Poynter		6 weeks		Operation.
Elizabeth A.Good	Diluted	22 days	15th	Relieved.
Rose George		36 days	15th	Relieved.
Lilian Babington	Citrated	30 days	14th	Cured.
Fanny Smith		20 days	16th	Cured
Rose Yates		76 days	29th	Operation
James Dowling		35 days		Operation
John Yates	Citrated	52 days		Relieved.
John Thomas Speak	Citrated then Allenbury's food	30 days	21st	Relieved.
Lizzie Green	Citrated	28 days	19th	Relieved.
Charles Hunt		36 days		Operation
Leonard Sigston		38 days		Operation.
Fred Hopkins		34 days		Operation.
John Hughes		30 days		Operation.
William Peters		32 days		Operation.
Elizabeth Caldicott		28 days	15th	Relieved.
Samuel Hutson	Raw Meat Juice	30 days	14th	Cured.
Emma Fletcher	Citrated	32 days		Cured.
Florrie Jones	Citrated	31 days		Operation.
Louise Hemms		28 days	14th	Relieved.

Not included in the above are 5 cases that died. Custard was also added to the "Middle" Diet in some cases. Under this treatment the patients gained in weight as much as 1 lb. in a week. The treatment adopted in the case of those that died was as follows:-

Case 1. Florrie Jones, admitted after an attack of haematemesis. Her condition was grave on admission. Salines per rectum every four hours and nutrient enemata were given every 6 hours. Sips of water were allowed from time to time. After 4 days Mr. Barling operated and performed a posterior gastro-enterotomy. Post-operative treatment was adopted, i.e., she was given continuous rectal saline for 24 hours, when it was stopped and albumin water one oz. 2 hourly given by the mouth; on the third day whey 2 oz. 2 hourly; 4th day whey 3 oz. 2 hourly; 5th, peptonised milk, 1 oz. hourly; 6th peptonised milk, 1 oz. with whey, 3 oz. hourly; 7th same as the 6th day; 8th day, diluted milk followed by custard. The patient was doing well until 16th day, when she had another and fatal haematemesis.

Case II. Charlotte Hardwick was admitted for pain and vomiting food. Her treatment consisted of citrated milk 3ß hourly for the first two days. On the 3rd day, ½ lb. raw meat juice peptonised and given as an enema; this nutrient enema alternated

6 hourly with egg and milk enema. All oral feeds were stopped, as pain and vomiting were increasing. These nutrients were continued until the 7th day when sips of water were given ½-hourly: the nutrients were still continued to the 9th day when feeds of citrated milk ℥ß hourly were given. The rectal nutrients were still continued. On the 10th day citrated milk ℥i hourly: salines were now stopped. The citrated milk was increased to ℥iii 2 hourly on the 11th day: nutrients still continued. On the 12th day citrated milk ℥iii 2 hourly alternated with Benger's Food ℥iii 2 hourly. On the 13th day oral feeds were stopped owing to vomiting and salines continued, as well as nutrients. Glucose solution ℥i was given 6 hourly per rectum instead of salines, which were given twice daily. The day following she suddenly collapsed from haemorrhage and died.

Case III. Arthur Horton was admitted after profuse haematemesis. The usual rectal enemata and salines were given in addition to adrenalin solution ℥ß orally, which was repeated alternate days. On the day of death, 4th after admission, horse serum ℥i was given, but a fatal haemorrhage caused collapse and death.

Case IV. William Reynaert was admitted after a sudden and profuse haematemesis. The same

treatment was adopted both before and after operation, as in the case of Florrie Jones, and reference to her case will show it. He died from haemorrhage.

Case V. Bridget Boland was admitted with complaint of being suddenly seized with severe pain in the abdomen. The treatment adopted was for the first six days milk and albumin water ℥i hourly. On 7th and 8th days milk ℥i only three hourly. On 9th and 10th milk ℥ii 2 hourly: whites of egg were added. From 10th to 26th day the diet was regulated carefully until solid food was reached. On the 39th day she was discharged relieved, only to be readmitted 7 weeks later with a recurrence of the same symptoms. She had rectal salines and nutrients for the first day, for on the second day she was operated on by Mr. Heaton. The post-operative treatment as indicated in Florrie Jones's case was used here. She got on to solid food, but was not long on them when symptoms of leakage from the ulcer appeared. A second operation was performed, ~~xx xxxxx~~ but the subphrenic abscess was not reached. She died with signs of portal pyaemia.

TREATMENT.

The conclusions from experiments by Bolton[47] are that a milk diet is the most suitable one,

because the epithelium grows over the base of an ulcer more rapidly than on any other diet. The base of a moderate sized ulcer is healed over in 20 days on a milk diet; on other diets it is still uncovered by that time. Milk does not stay long in the stomach or excite a copious gastric secretion. Milk diet and (b) rest in bed for three weeks at least are the two essentials in the cure of a gastric ulcer.

(c) Medicinal. The most universally employed drug is bismuth, which was recommended by Odier in 1786 as a panacea for spasm of the stomach. The subnitrate of bismuth has been largely advocated by Fleiner and Kussmaul and prescribed in 15 gm. doses. The method of these physicians is to wash out the stomach early in the morning and run into it a suspension mixture of 10 to 20 gms. of bismuth subnitrate in 200 c.c. of lukewarm water. The patient is told to lie in such a position that the ulcer comes into contact with the suspended bismuth. At the end of 5 or 10 minutes the tube is removed, but the patient still lies in the same position for half an hour. Where the passage of the stomach tube is contraindicated, the drinking of 10 gm.of bismuth subnitrate suspended in a glass of warm water is advised. Pariser (48) adopted a similar plan but advises the patient to drink on an empty stomach

drink 15 to 20 gms. of bismuth subnitrate stirred in water. He is told to lie down on his back for three quarters of an hour and then to take his breakfast. No toxic effects have been seen by any of these observers. Boas uses the carbonate. Fleiner explains the action of bismuth by assuming that the precipitate of bismuth protects the diseased portion of mucosa, and in this way prevents the corrosive action of the gastric juice.

In England bismuth is prescribed as a powder or in suspension, the dose being 10 to 15 grains. Bismuth is inclined to cause constipation which can be relieved by Carlsbad salts.

Bismuth is frequently prescribed along with the alkaline carbonates such as sodium bicarbonate and magnesium carbonate. The magnesium carbonate being an insoluble substance, except in the presence of an acid, will continue to neutralise the gastric acidity so long as it remains undissolved. The following is a suitable prescription

℞ Bismuthi Carbonates, gr. XX.
Magnesii Carbonates gr. X.
Sodii bicarbonates gr. V.
Aquae ad. ℥i.
Misce, fiat haustus. ℥i To be taken ½-hour before food, three times a day.

Cohnheim recommends 2 to 4 ounces of olive

oil in place of the large doses of bismuth and carried out by Fleiner's method.

In my cases bismuth as carbonate or subnitrate was prescribed always in suspension and in doses of gr. X. I gave along with it sodium bicarbonate or magnesium carbonate or gentian or rhubarb roots.

As many as 18 cases had the following mixture:

℞ Bis. Carb.
Sod. Bicarb. āā gr.X.
T. Cardamom Co. m.XV.
Mucilag.Trag q.S.
Aq ~~Lig~~.Chlorof. ad ℥i
M.et S. Ft.mist.
℥i. t.i.d., a.c.

Sometimes Ac. Hydrocyanic dil., would be given instead of T. Cardamom Co., especially if the pain was bad or vomiting became excessive.

In a few cases bismuth oxycarb. gr. XXX was given combined with Ac. hydrocyanic mπ No advantage seemed to be derived from its use.

Infusions of Gentian and Rhei were used as menstrua in cases of a more chronic nature than those above.

Another drug highly recommended is silver nitrate, which is an antacid and an astringent. Johnson was the first to recommend it, but used it for epilepsy and found that stomach symptoms disappeared. Gerhardt states that the drug acts particularly well in those cases where the pain is present when the stomach is empty. Boas recommends the drug in mild cases of ulcer or for those who cannot undergo a typical ulcer cure. Rosenheim administers the drug when there is hyperaesthesia of the stomach. Dreschfield has found that it stops the pain even better than morphia. It is given in pill form $\frac{1}{4}$ grain to a dose three times daily. Boas administers it in liquid form, beginning with $\frac{1}{2}$ grain three times daily and increasing it to one grain. Dreschfield has not seen any signs of argyria. The prescription in pill form is written as follows:-

℞ Argenti nitratis gr.1/3

Ungu. Kaolin q.S.

Ut.fiat pilula, one t.i.d.

I gave it in two cases only, after trying bismuth. The silver proved more beneficial than bismuth in these two cases. The alkaline carbonates are frequently administered alone, as they neutralise and prevent

hyperacidity and so lessen the irritation of the gastric secretions. They assist in promoting healing of the ulcer, and in the digestion of the food. Niemeyer was a great believer in the efficacy of the alkalies. The bicarbonates of soda and potash are used to lessen irritation and pain and are therefore useful sedatives. Ewald combines the alkalies with rhubarb and cane sugar. Riegel administers alkalies during or after food at the height of the hyperacidity. I invariably combined the alkalies with a salt of bismuth, the combination proving efficacious. All my gastric cases had Mist. Alba (an alkaline cathartic) every morning or twice a day. A ferruginous preparation, such as dried sulphate of iron is sometimes given, as it is capable of using up some of the excess of hydrochloric acid by forming an albuminate and a chloride compound. Anaemia is frequently combined with gastric ulcer so that the administration of iron serves a double purpose, one being the using up of excess of acid, the other relieving the anaemia. I prescribed iron in some form (usually as Ferrous sulphate) to as many as 17 of the patients. They all did extremely well and seemed to be benefited , especially after haematemesis. The patients seemed to recover more quickly with this treatment than with any other. The iron salt was usually combined with a saline cathartic to correct

any constipation which was always present whilst the iron remedied the loss of blood. One of my usual prescriptions was as follows:-

℞. Ferri. Sulph. gr iii
Mag. Sulph. ʒß
Ac. Sulph. dil. ♏v
Syr. Tolu. ♏xv
Aq. Chlorof. ad ʒi.
Ft. Mist.
Sig. ʒi. ter die.

After recovery the patients were advised to take Blaud's Pills one, two or three, three times daily. A purgative was frequently given which consisted of

℞. Ext. Cascar. Sagr. liq.
Ext. Glycyrrh. liq. āā ʒi.
Glycerol ʒß
Tinct. Nuc. Vom. ♏v.
Aq ad ʒß
Sig. ʒß b. d. s.

Medical Treatment: Chloretone (10 gr. in 10 oz. of water) has been recommended by Essex Wynter (49) to be given per rectum as a sedative in cases of recent

haematemesis, and the same drug by the mouth for pain and vomiting. He, as well as Schick,(50) believes in Extract of Belladonna ($\frac{1}{2}$ or $\frac{1}{4}$ gr. in pill), as it acts as a local anaesthetic and lessens gastric secretion and peristalsis. It can be prescribed as Atropine ($\frac{1}{2}$ to 1 mgram. hypo-dermically once or twice a day).

A new drug, Neutralon, a soluble aluminium silicate, has been used by Ehrmann(51) and Alexander, which is capable of combining gradually with hydrochloric acid to form the harmless insoluble silicic acid and aluminium chloride. Neutralon is a white, tasteless, odourless powder, insoluble in water. It was used in cases of gastric ulcer, hyperacidity and hypersecretion. In 18 cases of ulcer, 12 were successfully treated. The pain ceased after 3 to 5 days and tenderness disappeared in from 9 to 12 days. The best results were obtained after haemorrhage. They presume the silicate adheres better to a bleeding than a dry surface. He concludes that neutralon exercises a beneficial action in lessening gastric secretion. It acts slowly but the effect lasts as well as the alkali treatment. There are no intoxications from its use.

Normal horse serum has been recommended lately in the treatment of peptic ulcer: doses of 25 c.c. have been administered daily by the mouth. The benefit is

probably derived from its acid-fixing property. In one case only did I try horse serum and I gave it in 3i doses twice a day. Unfortunately the patient on whom it was tried died from haemorrhage arising out of acute ulceration. The patient was a man, Arthur Horton, who was particularly bad, dying four days after admission.

Chloroform in solution is advocated strongly by Stepp who administers it with bismuth every hour. I gave it either as spiritus chloroformi m.XV or Aqua, with good results. It lessened the vomiting. There are few cases of chronic ulcer that do not require occasional dosage of opium, especially to calm the severe attacks of pain and vomiting. A pill of the extract of opium ($\frac{1}{2}$ to 1 grain) may be given two or three times in 24 hours until the pain is relieved: or 5 to 15 minims of the liquor opii sedativus may be added to each dose of a bismuth mixture: hypodermic injection of morphine (1/6 - 1/4 gr.) may be given if great irritability and intolerance of the stomach are evident. It is of much use in cardialgia. I prescribed it only after a haematemesis and in doses of m.V of the liquor. It was necessary to repeat four hourly in some patients. I preferred to prescribe Ac. hydrocyanic dil. to reduce gastric irritability, which it was successful in doing. My cases responded well to it given in mii doses. Cocaine is sometimes called for to allay pain and

lessen the sensation of hunger. A tablespoonful of this mixture is useful.

℞ Cocainae hydrochloridi gr ii
Aquae laurocerasi ʒvi
Aquam ad ʒviii
Misce, fiat mistura. ʒß. s.o.s.

Internal and external use of ice has been found of use in allaying gastric irritability and relieving pain. When pain is due to hyperacidity, belladonna is frequently administered for its use in diminishing gastric secretion.

Treatment of symptoms and Complications.

Pain. The application of hot poultices, rest and regulation of diet are often sufficient to relieve it. The following drugs are used for its relief; bismuth, silver nitrate, morphine, atropine, and olive oil. Riegel states that the administration of morphine increases the secretion of hydrochloric acid.

Vomiting. No drugs are necessary unless it becomes very persistent. If the rest cure is properly carried out and the diet regulated, it is seldom necessary to give drugs unless alkalies. When it is very persistent, doses of dilute hydrocyanic acid or large doses of bismuth subnitrate often act well. Ice and cocaine are frequently used.

Haematemesis. The patient is immediately put in the recumbent posture and kept at absolute rest. All food by the mouth is stopped. He is not allowed to get up to pass urine or faeces. The thirst is relieved by washing out the mouth and allowing ice to melt on the tongue, or by giving rectal injections of warm water not exceeding 15 ounces at a time. Morphine hypodermically is given to keep the patient quiet. Ergotin is sometimes administered if the bleeding is profuse. Adrenalin chloride 1 in 1000 in doses of 10 to 30 drops is given to control the bleeding. Dreschfield has given turpentine, 20 to 30 minims, either in capsules or emulsion. Ewald has washed the stomach out with iced water with good results. An ice-bag is applied to the epigastrium. If the haemorrhage is very profuse and if collapse is imminent, stimulants should be given. The best of these is camphor given hypodermically. Enemata of wine broth are given per rectum as well as saline solution subcutaneously.

When the patient has recovered from the shock, it is advisable to start rectal enemata of peptonised foods 6 hourly alternated with rectal salines. Rectal feeding is to be persevered with at least for 4 to 6 days or longer, the patient the

while being in the recumbent posture. At the end of this time diluted cold milk by the mouth is started, or bouillon if milk is not tolerated. Albumin and yolk of egg in emulsion may be allowed also. During the second week the typical rest cure is adopted, that is, milk only, daily, hot poultices to the epigastrium, and Carlsbad salts in the morning. In the fourth week meat, certain vegetables and fruits are permitted if the objective and subjective conditions are good.

All my cases of recent or sudden haematemesis were treated on the lines just laid down, with the difference that I never administered ergotin or turpentine. Only in one case did I order turpentine to be administered. The case was the one that died from thrombosis of the portal vein. Of the nine cases of haematemesis, two died from the excessive haemorrhage. The arteries in each case were situated in the base of the ulcer and were of large size.

The treatment adopted after a haematemesis is best shewn in the case of John Yates, who was admitted with profuse haematemesis. Immediately he was put on to rectal salines, ½ pint 4 hourly: ice water to sip: hypod. morphia m.V. and adrenalin chloride m.XXX. The following day salines were continued and nutrients started every 6 hours. On the 2nd day, citrated milk ℥i hourly: salines were still continued. On the 3rd day, citrated milk ℥ii hourly with rectal salines. On the 4th day citrated milk

℥iii 2 hourly and 2 raw eggs. On the 5th day citrated milk ℥iv 4 hourly alternating with albumin water ℥iv. On the 6th day ½ lb. meat juice in 4 doses and continued for 3 days. On the 10th day custard, bread and butter were added. On the 12th day minced mutton was added and on the 15th day fish could be eaten.

(d) Surgical. Surgical intervention is usually resorted to after medical treatment has failed, or in the event of some complication arising during the course of the disease. Mayo Robson is of opinion that surgical measures should be adopted at a much earlier period than has hitherto been the custom. Surgeons emphasise this point, namely, that surgical cures are found in 90% of cases, whereas medical cures are variously given as 50% (Débove and Rémond), 25% (Paterson and Rhodes), 53% (Schultze), 42.6% (Russell), 85% (Welsh) and 74% (Leube). Again the mortality rate from surgical treatment is much lower and is diminishing every year. The surgical mortality is 5% (Mayo Robson and Moynihan) which contrasts with 22% (Stoll) as the medical mortality.

In any case the medical failures are high, which are variously estimated as 11% (Friedenwald), 44.7% (Russell): 47% (Schultze), 50 to 60% (Bulstrode), ~~74% (Leube), 80% (Welch)~~. From the above it will be observed that surgical treatment is very successful

and the cures from it are more likely to be permanent. Further, surgical treatment is self-preservation to the poorer classes, who form the bulk of patients with this complaint. Kreuzer, in Zurich, arrived at the following conclusions, (1) Secretions which were disturbed before were later normal. (2) Hyperacidity gave way to normal acidity or subacidity. (3) Reflux of bile and pancreatic juice into the stomach gave rise to no serious results. (4) Dilatation existing before operation had almost disappeared after.

The indications for surgical measures in gastric ulcer are: (1) chronic indolent ulcers resisting medical treatment and giving rise to the following symptoms:-
(a) Excessive vomiting and pain. (b) Recurrent haemorrhage. (c) Gastric dilatation, and hour-glass stomach. (d) Pyloric spasm and stenosis. (e) Perigastric adhesions. (f) Subphrenic abscess.
(2) Perforation, (3) Haematemesis in a case with mild dyspeptic symptoms over a long period.

It is in these chronic cases that the best results are obtained from operation. The pain and vomiting disappear and the patient rapidly gains in weight and is able to take ordinary food at the end of three weeks.

The treatment as carried out by the majority of surgeons consists in performing gastro-enterostomy with excision of the ulcer if possible.

The operation consists in fixing the highest portion of the jejunum to the stomach wall (usually posterior) and suturing the two, a small hole having been made in the stomach wall previously, to correspond to the lumen of the jejunum. In this way a fistula between the stomach and bowel is made. The ulcer, if it can be found, is removed or cauterised. This operation secures "physiological rest by means of drainage, thus allowing the ulcer to heal without being subjected to the irritation of the acid secretion, accumulation of food, or frequent stomach-movement." It also remedies "the hyperchlorhydria, relieves pyloric spasm, and diminishes gastric dilatation".

Gastroenterostomy was first performed by Doyen in 1892 for gastric ulcer, though Wölfler in 1881 performed it for a cancerous pylorus. Since that date the operation has found increasing favour with surgeons, until now it is one of the most frequent measures in surgical treatment.

The surgical treatment for (b) <u>persistent</u> recurrent haemorrhages, whether small or large, is gastroenterostomy, with excision of the ulcer which is chronic and indurated. Sometimes a (3) <u>haematemesis</u> may occur upon prolonged dyspepsia. In these cases also the ulcer is chronic and the tissues indurated and spontaneous healing more difficult. Here gastroenterostomy and excision of the ulcer are carried out.

If in acute cases a profuse and single haematemesis occurs, it is not advisable to operate, though much discussion has arisen on this point. It is estimated that 93% to 97% of these cases, if left alone and treated medically, recover.

(c) Gastric dilatation is treated surgically by removing the cause and the results. To do this, any of these methods is used, (1) resection of the pylorus, (2) Hemeke's pyloroplasty, (3) Gastro enterotomy, (4) Bircher's gastroplication, which reefs up the anterior wall and diminishes the size of the stomach.

Hour-Glass Stomach.is treated essentially by operative means, requiring a double operation. Many methods have been recommended, but the following are most frequently performed.

(1) Gastroplasty. This is similar to pyloroplasty and is performed so that on the site of the constriction a longitudinal incision is made through the anterior wall and it is then united transversely.

(2) Gastro-anastomosis is similar to gastro-enterotomy, but a connection between the two bags is made.

(3) Gastro-enterostomy is of little use by itself and in order to be beneficial, it should be duuble.

(4) Partial gastrectomy. In this operation the total area of disease is removed and the healthy ends of the stomach are united.

Mayo Robson reported 19 cases of simple hour-glass stomach, in 11 of which he did gastroplasty, with recoveries in all. In 6 others a posterior gastroenterostomy was performed, followed by complete recovery. Moynihan performed gastroplasty in 7 cases with good results, and gastroenterostomy in 7 cases which were greatly relieved.

(d) Pyloric spasm and stenosis This is treated by gastroenterostomy, pylorectomy, pyloroplasty or forcible dilatation of the pylorus. Posterior gastroenterostomy has the advantage of being brief and rapid, and is usually followed by brilliant results.

(e) Perigastric adhesions.are a complication of chronic gastric ulcer, and are the result of gastric ulcer with adhesions to the parietes or neighbouring organs. It is relieved by gastrolysis or by operation of posterior gastroenterostomy. Severance of the adhesions has been successfully reported by V. Haeker, Lauenstein, and V. Mickulicz. Klemperer has attempted to get rid of such fibrous bands by hypodermic injections of fibrolysin.

Of the cases falling under the above groups, there were eight that were submitted to operation. Four were operated on for recurrent haemorrhage and

four for chronicity of ulceration and two for gastrectasis. Some of these cases displayed adhesions to other organs. In all these cases posterior gastroenterostomy was performed. The four cases operated on for chronicity of ulceration were (a) Charles Hunt, (b) Leonard Sigston, (c) Fred Hopkins, (d) Lizzie Green.

(a) Charles Hunt, aged 39, had been suffering for eight years from chronic indigestion and was operated on by Mr. Heaton. He found an old indurated ulcer on the lesser curvature about 2 inches from the pylorus. The pylorus was closed with a purse-string suture and a posterior gastroenterostomy was done. He made a successful recovery.

(b) Leonard Sigston, aged 36, had suffered from chronic indigestion for five years. At the operation a hard mass was felt surrounding the pylorus and extending one inch from it. A posterior gastroenterostomy was performed by Mr. Heaton.

(c) Fred Hopkins had had symptoms of indigestion for seven years. He was operated on by Mr. Lucas who found a large indurated ulcer situated on the lesser curvature and extending into the lumen of his stomach. It was impossible to excise it. A posterior gastroenterostomy was performed.

(d) Lizzie Green had had indigestion for five years. She was operated on by Mr. Haslam who found a thickened mass in the lesser curvature near the pylorus. A porterior gastroenterostomy was performed.

Four cases presented recurrent attacks of haematemesis, in all of whom a posterior gastroenterostomy was done. Unfortunately two died, not as the result of the operation but from another attack of haematemesis

Case 1. James Dowling had an attack of indigestion for two years with several attacks of haematemesis during that period. Mr. Haslam found at the operation a thick indurated mass near the pylorus along the lesser curvature; so a posterior gastroenterostomy was done. He made a good recovery.

Case 11. Rose Yates had suffered from indigestion and attacks of haematemesis for five years. She was admitted during an attack of haematemesis. A month later Mr. Heaton operated and found the stomach tied down to the liver and pancreas and an ulcer was situated in the posterior wall. He performed a posterior gastroenterostomy and the patient was discharged cured.

Case 111 Florrie Jones had been an out-patient suffering from indigestion and occasional vomiting of blood. She was admitted immediately

after an attack of haematemesis. Mr. Barling operated 14 days later and found a large ulcer on the lesser curvature near the pylorus and extended on to the anterior as well as posterior walls of the stomach. A posterior gastroenterostomy was done. Sixteen days later she died from a suppurative peritonitis.

Case IV. William Reynaert had indigestion for 10 days, when on the day of admission he had a very severe attack of haematemesis. He had two similar attacks of haematemesis, so it was decided to operate. Mr.Heaton operated on the 7th day after admission and found the stomach dilated and adhesions stretching across lesser sac between the posterior surface to the stomach bed. Two ulcers were found in the posterior aspect near the lesser curvature. Posterior gastroenterostomy was done. Six days later he had a violent attack of haematemesis which proved fatal.

Two cases were operated upon for pyloric stenosis and gastrectasis.

Case I William Peters had relapses of indigestion for 8 years. On admission his stomach was found to reach half way between the umbilicus and fubis and succussion easily obtained. Mr. Heaton operated and found the pylorus was stenosed and hardened with a double ring of contraction. The stomach was enormously dilated and thin-walled.

Posterior gastroenterostomy was done. The patient made a good recovery.

Case II. John Hughes had indigestion for 3 years. On admission the stomach reached the umbilicus and succussion obtained. Mr. Heaton operated and found an indurated thickened and puckered ulcer situated on the posterior aspect near pylorus. The stomach was considerably dilated. A posterior gastroenterostomy was done. He made a very satisfactory recovery.

(f) Subphrenic Abscess. When a chronic ulcer has begun to leak with escape of stomach contents into the peritoneal cavity, the sooner the patient is operated on the better the chance of recovery. I had two cases both operated upon, one of which died.

Case 1 Bridget Boland had been seized with abdominal symptoms about one month before admission; then the symptoms recurred suddenly on day of admission. She had suffered from indigestion for 5 years. There was no rigidity, but epigastric and dorsal tenderness was present: liver dulness was not obliterated. The persistence of pain in the abdomen was marked; the abdomen was resistant and the temperature elevated for three days. These symptoms subsided andthe patient was discharged, only to be readmitted after seven weeks with a recurrence of the symptoms. This time the abdomen was markedly resistant, rigid and

tender: the temperature was 95° F. and the pulse 86 per minute. On the following day Mr. Heaton operated and found a large thickened area in the lesser curvature of the stomach with fresh adhesions to the liver. The abdomen was closed but no gastro-enterostomy was done. Six weeks later she died from a subphrenic abscess formed from the ulcer that had been slowly leaking.

Case II. Robert Poynter had been in hospital with haematemesis 2 years before and was readmitted with the same symptoms. Suddenly the ulcer began to leak, the abdomen became distended and tympanitic and liver dulness was obliterated: the temperature was 99° F. and the pulse 100 per minute. He was operated on by Mr. Clayton Greene, who found adhesions of the stomach to the transverse colon, and the peritoneal cavity contained pus. The abscess was drained and the patient made a good recovery. Later a posterior gastro-enterostomy was done. Both of these cases have been fully described previously.

(2) Perforation. Spontaneous healing can only occur if the stomach is completely empty at the time of perforation. In this case circumscribed peritonitis develops, ultimately leading to the formation of adhesions.. If the stomach is full at the time of perforation, operation should be performed as soon as possible. The later the surgeon operates, the smaller the chances

of recovery. Previous to operation, the mortality was 95%: now the mortality is reduced in direct proportion to the stage in which the operation takes place. If within 12 hours of rupture, the mortality is 25% to 28%: in from 12 to 24 hours, 63%: and after 24 hours 86% to 100 %. The operation should not be performed until the first shock has passed off, that is, about 10 hours after. These statistics are taken from Weir and Foot's (52) analysis of 169 cases.

The operation performed is either that of excision of the ulcer or a folding - in of the ulcer with the application of a continuous suture. Sometimes a gastroenterostomy is performed, which allows complete rest to the ulcerated area.

I shall mention one case illustrative of this condition. William Lewis was suddenly seized with abdominal pain which doubled him up. He gave a history of chronic indigestion. Mr. Barnes operated and found stomach contents in the peritoneal cavity. A larger ulcer, one inch in diameter, was found on the lesser curvature about 2½ inches from the pylorus: the edges were cleanly cut. The ulcer was directly closed by silk sutures andthe stomach wall then stitched over it. He made a very good recovery.

The after treatment of cases of cured gastric ulcer must be conducted on general principles. The anaemia must be treated with mild preparatives of iron, the dyspepsia by careful and cautious feeding.

The frequent coexistence of constipation should be treated by enemata of salt and water or by a dose of Carlsbad salts in the morning.

Finally, when in doubt whether a case is one of gastric ulcer or not, especially if in a young chlorotic girl, it is best to treat the case as if it were one of ulceration. It is in this class of patient that perforating ulcer of the stomach is apt to run a latent course and to end suddenly in perforation or be attended with sudden and profuse haematemesis.

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